Generate Collection

L4: Entry 2 of 6

File: USPT

May 2, 2000

DOCUMENT-IDENTIFIER: US 6057125 A TITLE: Clock gene and gene product

DEPU:

Vitatema, M. H., D. P. King, A.-M. Chang, J. M. Kornhauser, P. L. Lowrey, J. D. McDonald, W. F. Dove, L. P. Pinto, F. W. Turek, J. S. Takahashi. 1994. <u>Mutagenesis and mapping</u> of a mouse gene, Clock, essential for circadian behavior. Science 264:719-725

DEPU:

Vitatema, M. H., D. P. King, A.-M. Chang, J. M. Kornhauser, P. L. Lowrey, J. D. McDonald, W. F. Dove, L. P. Pinto, F. W. Turek, J. S. Takahashi. 1994. <u>Mutagenesis and mapping</u> of a mouse gene, Clock, essential for circadian behavior. Science 264:719-725



US Patents Full-Text Database US Pre-Grant Publication Full-Text Database JPO Abstracts Database Database: EPO Abstracts Database Derwent World Patents Index **IBM Technical Disclosure Bulletins** Term: Display: Documents in Display Format: CIT Starting with Number 1 Generate: O Hit List O Hit Count O Image Search Clear Help Logout Interrupt Main Menu Show S Numbers Edit S Numbers Preferences

Search History

Today's Date: 4/2/2001

DB Name	Query	Hit Count	Set Name
USPT,PGPB,JPAB,EPAB,DWPI	congenic and 11	0	<u>L7</u>
USPT,PGPB,JPAB,EPAB,DWPI	shedlovsky-a\$.in.	4	<u>L6</u>
USPT,PGPB,JPAB,EPAB,DWPI	dove-w\$.in.	7	<u>L5</u>
USPT,PGPB,JPAB,EPAB,DWPI	mutagenesis adj1 mapping	6	<u>L4</u>
USPT,PGPB,JPAB,EPAB,DWPI	12 same mutagenesis	3	<u>L3</u>
USPT,PGPB,JPAB,EPAB,DWPI	congenic	254	<u>L2</u>
USPT,PGPB,JPAB,EPAB,DWPI	modifier adj1 (locus or loci)	9	<u>L1</u>

```
L5
     ANSWER 4 OF 4 MEDLINE
ΑN
     1998207250
                    MEDLINE
DN
     98207250
ΤI
     A high-resolution genetic map of the nervous locus on mouse
     chromosome 8.
     De Jager P L; Harvey D; Polydorides A D; Zuo J; Heintz N
IΙΔ
     Howard Hughes Medical Institute, Laboratory of Molecular Biology,
CS
     Rockefeller University, New York, New York 10021, USA.
NC
     GM07739 (NIGMS)
SO
     GENOMICS, (1998 Mar 15) 48 (3) 346-53.
     Journal code: GEN. ISSN: 0888-7543.
CY
     United States
DT
     Journal; Article; (JOURNAL ARTICLE)
LA
     English
FS
     Priority Journals
EM
     199808
     19980802
EW
    The nervous (nr) mutant mouse displays two gross recessive traits: both
AB
an
    exaggeration of juvenile hyperactivity and a pronounced ataxia become
    apparent during the third and fourth postnatal weeks. Using an
    intersubspecific intercross, we have established a high-resolution
```

exaggeration of juvenile hyperactivity and a pronounced ataxia become apparent during the third and fourth postnatal weeks. Using an intersubspecific intercross, we have established a high-resolution map of a segment of mouse chromosome 8 that places the nr locus in a genomic segment defined by D8Rckl on the centromeric end and D8Mit3 on the telomeric end. This map position places the nr locus within the BALB/cGr congenic region of the C3HeB/ FeJ-nr strain, confirming the accuracy of our study. We used this map position to identify and evaluate three genes-ankyrin 1, cortexin, and farnesyltransferase-as candidates for the nr gene. These three genes were eliminated from consideration but allowed us to establish the

of synteny between the region containing the nr locus and a segment of the

short arm of human chromosome 8 (8p21-p11.2). Finally, the incomplete penetrance of the nr phenotype led us to perform a screen for **modifier loci**, and we present evidence that such a nervous **modifier locus** may exist on mouse chromosome 5.

L30

```
(FILE 'HOME' ENTERED AT 16:41:46 ON 02 APR 2001)
      FILE 'MEDLINE' ENTERED AT 16:41:53 ON 02 APR 2001
L1
               0 S CONGENIC AND MUTAGENESIS MAPPING/AB, BI
L2
               3 S MUTAGENESIS MAPPING/AB, BI
L3
             108 S MODIFIER LOCUS OR MODIFIER LOCI/AB, BI
L4
              10 S L3 AND CONGEN?/AB, BI
L5
               4 S L4 AND MAP?/AB, BI
L6
               0 S L3 AND L2
     FILE 'MEDLINE, EMBASE, BIOSIS, INPADOC, CAPLUS' ENTERED AT 16:46:21 ON
02
     APR 2001
              0 S L1
L7
L8
              42 S L2
L9
             442 S L3
L10
             62 S L4
            484 S L8 OR L9 OR L10
L11
L12
             39 S L11 AND BACKCROSS?/AB, BI
L13
             13 DUP REM L12 (26 DUPLICATES REMOVED)
                E DOVE WILLIAM F/AU
L14
            133 S E2-E3
L15
             12 S L14 AND L3
L16
              7 DUP REM L15 (5 DUPLICATES REMOVED)
L17
              0 S L14 AND L2
L18
              0 S L14 AND L10
                E SHEDLOVSKY ALEXANDRA/AU
L19
             88 S E1-E4
L20
             12 S L11 AND (L19 OR L14)
L21
              7 DUP REM L20 (5 DUPLICATES REMOVED)
L22
           5586 S ETHYLNITROSOUREA/AB, BI
L23
           1788 S L22 AND MUTAGEN?/AB, BI
L24
              1 S L23 AND L9
L25
              7 S L23 AND BACKCROSS?/AB, BI
L26
              4 DUP REM L25 (3 DUPLICATES REMOVED)
L27
             39 S L9 AND BACKCROSS?/AB, BI
L28 ·
              2 S L27 AND MUTAGEN?/AB, BI
L29
              2 DUP REM L28 (0 DUPLICATES REMOVED)
```

13 DUP REM L27 (26 DUPLICATES REMOVED)

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(FILE 'HOME' ENTERED AT 16:41:46 ON 02 APR 2001)
     FILE 'MEDLINE' ENTERED AT 16:41:53 ON 02 APR 2001
               0 S CONGENIC AND MUTAGENESIS MAPPING/AB, BI
L1
L2
               3 S MUTAGENESIS MAPPING/AB, BI
            108 S MODIFIER LOCUS OR MODIFIER LOCI/AB, BI
L3
L4
             10 S L3 AND CONGEN?/AB, BI
L5
               4 S L4 AND MAP?/AB, BI
L6
               0 S L3 AND L2
     FILE 'MEDLINE, EMBASE, BIOSIS, INPADOC, CAPLUS' ENTERED AT 16:46:21 ON
02
     APR 2001
L7
              0 S L1
^{\text{L8}}
             42 S L2
L9
            442 S L3
L10
             62 S L4
L11
            484 S L8 OR L9 OR L10
L12
             39 S L11 AND BACKCROSS?/AB, BI
L13
             13 DUP REM L12 (26 DUPLICATES REMOVED)
                E DOVE WILLIAM F/AU
L14
            133 S E2-E3
L15
             12 S L14 AND L3
L16
              7 DUP REM L15 (5 DUPLICATES REMOVED)
L17
              0 S L14 AND L2
=> s 114 and 110
             0 L14 AND L10
=> e shedlovsky alexandra/au
E1
            61
                   SHEDLOVSKY A/AU
E2
                   SHEDLOVSKY A E/AU
            3
E3
            23 --> SHEDLOVSKY ALEXANDRA/AU
E4
             1
                   SHEDLOVSKY ALEXANDRA J/AU
E5
            8
                   SHEDLOVSKY J P/AU
                   SHEDLOVSKY JULIAN P/AU
E6
             5
Ε7
            3
                   SHEDLOVSKY LEO/AU
E8
            5
                   SHEDLOVSKY THEODORE/AU
E9
             3
                   SHEDLOW A M/AU
E10
             1
                  SHEDLOW ALEXANDRA/AU
E11
                   SHEDLOW ALEXANDRA M/AU
E12
             1
                   SHEDLOW ALEXANDRA MARY/AU
=> s e1-e4
L19
            88 ("SHEDLOVSKY A"/AU OR "SHEDLOVSKY A E"/AU OR "SHEDLOVSKY
ALEXAND
               RA"/AU OR "SHEDLOVSKY ALEXANDRA J"/AU)
=> s 111 and (119 or 114)
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12 L11 AND (L19 OR L14)

L20

FILE HOME' ENTERED AT 16:41:46 ON 02 APR 200

=> file medline

TOTAL 0.15 SINCE FILE SESSION 0.15 ENTRY FULL ESTIMATED COST COST IN U.S. DOLLARS

FILE 'MEDLINE' ENTERED AT 16:41:53 ON 02 APR 2001

FILE LAST UPDATED: 22 MAR 2001 (20010322/UP). FILE COVERS 1958 TO DATE.

database. These records have an Entry Date and Update Date of MEDLINE now contains new records from the former NLM HEALTH STAR 20010223 MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2001 vocabulary. Enter HELP THESAURUS for details.

The OLDMEDLINE file segment now contains data from 1958 Enter HELP CONTENT for details. Left, right, and simultaneous left and right truncation are available in

Basic Index. See HELP SFIELDS for details

THIS FILE CONTAINS CAS REGISTRY NUMBERS FOR EASY SUBSTANCE IDENTIFICATION AND ACCURATE

regular updates to the file are not in place. As soon as NLM makes (20010322/UP). NLM is still in the process of preparing data. MEDLINE has been updated with new records for the 2001 Therefore,

=> s congenic and mutagenesis mapping/ab,bi

regular updates available, we will process the update

57235 MUTAGENESIS/BI 143268 MAPPING/BI 3481 CONGENIC 5621341 AB/FA

((MUTAGENESIS(W)MAPPING)/BI (L) AB/FĄ) 2 MUTAGENESIS MAPPING/AB S7235 MUTAGENESIS/BI 143268 MAPPING/BI

3 MUTAGENESIS MAPPING/BI

((MUTAGENESIS(W)MAPPING)/BI) 0 CONGENIC AND MUTAGENESIS MAPPING/AB,BI _

57235 MUTAGENESIS/BI

=> s mutagenesis mapping/ab,bi

143268 MAPPING/BI 5621341 AB/FA

((MUTAGENESIS(W)MAPPING)/BI (L) AB/FA) 2 MUTAGENESIS MAPPING/AB

57235 MUTAGENESIS/BI 43268 MAPPING/BI

((MUTAGENESIS(W)MAPPING)/BI) 3 MUTAGENESIS MAPPING/AB,BI 3 MUTAGENESIS MAPPING/BI 1.2

=> d 1. bib ab

YOU HAVE REQUESTED DATA FROM 3 ANSWERS CONTINUE? Y/(N):y

L2 ANSWER I OF 3 MEDLINE
AN 1998035455 MEDLINE
DN 98035455
TI The crystal structure of vascular endothelial growth factor (VEGF)

to 1.93 A resolution: multiple copy flexibility and receptor binding.

AU Muller Y A; Christinger H W; Keyt B A; de Vos A M CS Department of Protein Engineering, Genentech, Inc., South San

CA 94080, USA.

SO STRUCTURE, (1997 Oct 15) 5 (10) 1325-38. Journal code: B31, ISSN: 0969-2126.

ENGLAND: United Kingdom ς

Journal; Article; (JOURNAL ARTICLE)

English

FS Priority Journals EM 199803

AB BACKGROUND: Vascular endothelial growth factor (VEGF) is an endothelial

cell-specific angiogenic and vasculogenic mitogen. VEGF also plays a role

development of VEGF antagonists, which prevent the interaction of in pathogenic vascularization which is associated with a number of clinical disorders, including cancer and rheumatoid arthritis. The

with its receptor, may be important for the treatment of such disorders

superfamily, showing greatest similarity to platelet-derived growth VEGF is a homodimeric member of the cystine knot growth factor

(PDGF). VEGF binds to two different tyrosine kinase receptors,

domain receptor (KDR) and Fms-like tyrosine kinase 1 (Fit-1), and of VEGF homologs are known with distinct patterns of specificity

same receptors. The structure of VEGF will help define the

receptor-binding site, and shed light on the differences in specificity and cross-reactivity among the VEGF homologs. RESULTS: We

resolution in a triclinic space group containing eight monomers in the crystal structure of the receptor-binding domain of VEGF at

asymmetric unit. Superposition of the eight copies of VEGF shows

beta-sheet core regions of the monomers are very similar, with

greater differences in most loop regions. For one loop, the different ***Mutagenesis*** ***mapping*** shows that this loop is copies represent different snapshots of a concerted motion

receptor-binding site of VEGF, CONCLUSIONS: A comparison of part of the

independent copies of VEGF in the asymmetric unit indicates the

conformational space sampled by the protein in solution; the root

square differences observed are similar to those seen in ensembles highest precision NMR structures. Mapping the receptor-binding

determinants on a multiple sequence alignment of VEGF homologs, suggests

sequence variation and changes in the flexibility of binding loops the differences in specificity towards KDR and Flt-1 may derive

structure can also be used to predict possible receptor-binding determinants for related cystine knot growth factors, such as PIOGF

ANSWER 2 OF 3 MEDLINE

AN 83223569 MEDLINE DN 83223569

mutagenesis ***mapping*** of a recombinant cDNA T1 Localization of a Plasmodium surface antigen epitope by Tn5

AU Lupski J R; Ozaki L S; Ellis J; Godson G N SO SCIENCE, (1983 Jun 17) 220 (4603) 1285-8.

Journal code: UJ7, ISSN: 0036-8075. CY United States Journal; Article; (JOURNAL ARTICLE) English

Priority Journals; Cancer Journals

AB A recombinant complementary DNA clone from Plasmodium

beta-lactamase fusion polypeptide in Escherichia coli that reacts knowlesi makes a

monoclonal antibody to a Plasmodium surface antigen. An epitope

surface antigen was localized by transposon Tn5 ***mutagenesis***

Mapping of melanoma ***modifier*** ***loci*** EM 200102 AB Transgenic mice carrying the RET oncogene under the control of metallothionein promoter exhibit severe pigmentation of the whole three melanoma ***modifier*** ***loci***, on chromosome ***locus*** , we intestinal tumors in ENU-treated Min/+ mice ***maps*** to a ***Mapping*** of Melm loci and of five the resistance is due to a linked ***modifier*** ***locus*** melanocytic tumors. The genetic background influences melanoma RET mice on a C57BL/6 genetic background (N3/RET mice), we generated several lines of mice carrying different regions of the incidence and increased latency of melanocytic tumors, whereas and Melm2) and chromosome 11 (Melm3), that are linked with additional regions on chromosomes 6, 8, 9, 12, and 13 indicated AU Dragani T A; Peissel B; Zanesi N; Aloisi A; Dai Y; Kato M; ***congenic*** interval. We have found that resistance to 4-cM interval that includes the ROSA26 LacZ-neoR insertion. resistance to turnor development is due to either the ROSA26 SO JAPANESE JOURNAL OF CANCER RESEARCH, (2000 linkage in (N3/RETxBALB/c)xN3/RET backcross mice. We in RET mice, founder mice crossed with BALB/c mice show CS Department of Experimental Oncology, Istituto Nazionale C57BL/6 mice show the opposite effect. Using partially ***locus*** Venezian Milan, Italy.. dragani@istitutotumori.mi.it further ***map*** the ***modifier*** DT Journal; Article; (JOURNAL ARTICLE) Journal code: HBA, ISSN: 0910-5050. very tightly linked ***modifier*** L5 ANSWER 2 OF 4 MEDLINE AN 2001101547 MEDLINE DN 20545364 incidence and latency. Nov) 91 (11) 1142-7. FS Priority Journals transgenic mice. Nakashima I ***congenic*** Furnori, Via G. studied genetic ***mapped*** early melanoma Therefore, the mammary and insertion or a LA English development CY Japan progeny of Suzuki H. decreased in RET skin and the effect. Each of these ***congenic*** lines carries approximately CY United States
DT Journal: Article: (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200102
AB B6.129S7-Ctrosa26 (B6.R26) mice carry a LacZ-neoR insertion from two independently derived ***congenic*** lines show this of 129-derived DNA flanking the insertion, raising the possibility TI ROSA26 mice carry a modifier of Min-induced mammary and intestinal tumor (Chr) 6, made by promoter trapping with 129 ES cells. Female ApcMin/+ (B6Min/+) mice are highly susceptible to intestinal intestinal tumors after ENU treatment than do B6 Min/+ mice. MAMMALIAN GENOME, (2000 Dec) 11 (12) 1058-62. (ENU). However, B6.R26/+ Min/+ females develop fewer YOU HAVE REQUESTED DATA FROM 4 ANSWERS. the induction of mammary tumors after treatment with AU Kohlhepp R L; Hegge L F; Nett J E; Moser A R CS Department of Human Oncology, University of Wisconsin-Madison 53792, USA.
NC CA64843 (NCI)
SO MAMMALIAN GENONE; (2000 Dec) 11 (12) 10 L3 AND CONGEN?/AB.BI Journal code: BES, ISSN: 0938-8990. (CONGEN?/BI (L) AB/FA) ANSWER 1 OF 4 MEDLINE (MAP?/BI (L) AB/FA) 4 L4 AND MAP?/AB,BI AN 2001122351 MEDLINE 146823 CONGEN7/BI 62849 CONGEN?/AB 146823 CONGEN?/BI 97484 MAP?/AB => s l4 and map?/ab,bi CONTINUE? Y/(N):y 192862 MAP?/BJ 192862 MAP?/BI 5621341 AB/FA 5621341 AB/FA development. on Chromosome ethylnitrosourea DN 21015401 => d 1- bib ab B6.R26/+ mice mammary and turnors and to C57BL/6J modifier 20 cM 7 LS

AB Nitrosoguanidine-induced mutations occur at higher frequencies

replication region than at other nonreplicating regions of the

SO JOURNAL OF BACTERIOLOGY, (1981 Jul) 147 (1) 176-80.

DT Journal; Article; (JOURNAL ARTICLE) Journal code: HH3. ISSN: 0021-9193.

CY United States

Priority Journals

4 FS.

EM 198110 English

AU Woodley CL; Baldwin J N; Greenberg J

Mycobacterium tuberculosis genes.

of mapping of

II Nitrosoguanidine sequential ***mutagenesis***

ANSWER 3 OF 3 MEDLINE

L2 ANSWER 3 OF 3 MEDI AN 81215331 MEDLINE

DN 81215331

the farthest 5' insert into the complementary DNA portion of the ***mapping*** of the complementary DNA clone. The Tn5

gene, giving the shortest truncated protein that maintained the bind monoclonal antibody, defined the location of the epitope. drug resistance. Use of M. tuberculosis provided a 10-h replication

were used to determine the order of replication for 10 genes Cultures of Mycobacterium tuberculosis synchronized with

phenylethanol

replication. The direction of chromosome replication could not be

determined, but this study indicated no pause between rounds of

deoxyribonucleic acid replication in a rich medium.

=> s modifier locus or modifier loci/ab,bi

(MODIFIER(W)LOCUS)

2973 MODIFIER/BI

29848 LOCI/BI 5621341 AB/FA

67 MODIFIER LOCUS

2973 MODIFIER

57107 LOCUS

((MODIFIER(W)LOCIYBI)
108 MODIFIER LOCUAB,BI

S

=> s 13 and congen?/ab,bi

53 MODIFIER LOCI/BI

((MODIFIER(W)LOCI)BI (L) AB/FA)

2973 MODIFIER/BI

29848 LOCI/BI

51 MODIFIER LOCI/AB

with good resolution because of the slow rate of deoxyribonucleic

BALB/c susceptibility/ ***modifier*** ***locus*** , Petrl, is a genomic segment defined by D8Rck1 on the centromeric end and the telomeric end. This ***map*** position places the nr locus famesyltransferase-as candidates for the nr gene. These three genes traditionally susceptible BALB/cAn strain. Biological assays of the p16(INK4a) and p19(ARF) alleles from BALB/c and DBA/2 TI A high-resolution genetic ***map*** of the nervous locus on alleles displayed similar potencies in both assays. We propose that pristane-induced plasma cell tumors over a shorter latency period ***map*** of a segment of mouse chromosome 8 that places intersubspecific intercross, we have established a high-resolution confirming the accuracy of our study. We used this ***map*** LA English FS Priority Journals EM 199808 EW 19980802 AB The nervous (nr) mutant mouse displays two gross recessive the BALB/cGr ***congenic*** region of the C3HeB/ FeJ-nr exaggeration of juvenile hyperactivity and a pronounced ataxia apparent during the third and fourth postnatal weeks. Using an AU De Jager P L; Harvey D; Polydorides A D; Zuo J; Heintz N CS Howard Hughes Medical Institute, Laboratory of Molecular Biology. eliminated from consideration but allowed us to establish the inducing growth arrest of mouse plasmacytoma cell lines and to identify and evaluate three genes-ankyrin 1, cortexin, and Rockefeller University, New York, New York 10021, USA. ras-induced transformation of NIH 3T3 cells, while the two BALB/c p16(INK4a) allele was less active than its DBA/2 NC GM07739 (NIGMS) SO GENOMICS, (1998 Mar 15) 48 (3) 346-53. Journal code: GEN. ISSN: 0888-7543. Journal; Article; (JOURNAL ARTICLE) "efficiency" allele of the p16(INK4a) gene L5 ANSWER 4 OF 4 MEDLINE AN 1998207250 MEDLINE CY United States DT Journal; Articl chromosome 8. the nr locus in counterpart in traits: both an preventing Cdkn2a, which encodes p16(INK4a) and p19(ARF), and the coding strains are genetically resistant to the plasmacytomagenic effects of ***mapped*** to a 5.7-centimorgan (cM) chromosomal region interval. Concomitantly, resistant C57BL/6 mice, from which both Institute, National Institutes of Health, Bethesda, Maryland 20892. introgressively backcrossed to the susceptible BALB/c strain. The AU Zhang S L; DuBois W; Ramsay E S; Bliskovski V; Morse H C; pristane. In this model system for human B-cell neoplasia, one of for the BALB/c p16(INK4a) and p19(ARF) alleles were found to resultant C.DAG-Pctr1 Cdkn2a D4Mit15 ***congenic*** was TI Efficiency alleles of the Petr1 ***modifier*** ***locus*** D4Mit15 from a resistant strain (BALB/cDAG) carrying DBA/2 to plasmacytomagenesis than BALB/c, thus narrowing Petr1 to a imbalance in N3/RET mice, with a significant excess of BALB/c SO MOLECULAR AND CELLULAR BIOLOGY, (2001 Jan) 21 polymorphic with respect to their resistant Petr1 counterparts in complex genetic trait involving multiple loci, while DBA/2 and CS Laboratory of Genetics, Division of Basic Sciences, National products of the Cdkn2a gene have been eliminated, developed BALB/c susceptibility and ***modifier*** ***loci***, and C57BL/6 mice (45). In the present study, alleles of Petrl, The susceptibility of BALB/c mice to pristane-induced suggesting the presence of additional putative melanoma Journal; Article; (JOURNAL ARTICLE) L; Vass W C; DePinho R A; Mock B A Journal code: NGY, ISSN: 0270-7306. ***loci*** on these chromosomes. ANSWER 3 OF 4 MEDLINE 2001089278 MEDLINE plasmacytoma susceptibility. Priority Journals CY United States plasmacytomas is a 20565764 EM 200101 English that included Pctr1, was (1) 310-8

nervous ***modifier*** ***locus*** may exist on mouse ***modifier*** ***loci*** , and we present evidence that short arm of human chromosome 8 (8p21-p11.2). Finally, the PROCESSING COMPLETED FOR L12 L13 13 DUP REM L12 (26 DUPLICATES REMOVED) penetrance of the nr phenotype led us to perform a screen for YOU HAVE REQUESTED DATA FROM 13 ANSWERS CONTINUE? Y(N);y 'AB' IS NOT A VALID FIELD CODE L12 39 L11 AND BACKCROSS#AB,BI => file medline embase biosis inpadoc caplus 'AB' IS NOT A VALID FIELD CODE 484 L8 OR L9 OR L10 => s 111 and backcross?/ab,bi 0 L3 AND L2 => s 18 or 19 or 110 62 1.4 => dup rem 112 segment of the 42 [.2 => s l3 and l2 017 => d 1- bib ab chromosome => s 12 => s 14 => s]] => s l3 Ξ ያ

of synteny between the region containing the nr locus and a

KG, KZ, MD, IN, IS, strains are genetically resistant to the plasmacytomagenic effects of respect to their resistant Pctr1 counterparts in DBA/2 and C57BL/6 and NIH/Ola. These results, therefore, illustrate the general use of Institute, National Institutes of Health, Bethesda, Maryland 20892, DUPLICATE interval. Concomitantly, resistant C57BL/6 mice, from which both Zhang S L; DuBois W; Ramsay E S; Bliskovski V; Morse H C; ***|ocus*** pristane. In this model system for human B-cell neoplasia, one of (45). In the present study, alleles of Pctr1, Cdkn2a, and D4Mit15 interspecific crosses between Mus musculus and Mus spretus for SO MOLECULAR AND CELLULAR BIOLOGY, (2001 Jan) 21 detection of strong genetic interactions between tumor modifier complex genetic trait involving multiple loci, while DBA/2 and L; Vass W C; DePinho R A; Mock B A CS Laboratory of Genetics, Division of Basic Sciences, National resistant strain (BALB/cDAG) carrying DBA/2 chromatin were C.DAG-Petr1 Cdkn2a D4Mit15 ***congenic*** was more plasmacytomagenesis than BALB/c, thus narrowing Petr1 to a BALB/c p16(INK4a) and p19(ARF) alleles were found to be ***backcrossed*** to the susceptible BALB/c strain. The mapped to a 5.7-centimorgan (cM) chromosomal region that which encodes p16(INK4a) and p19(ARF), and the coding LA English
FS Priority Journals
EM 200101
AB The susceptibility of BALB/c mice to pristane-induced ***modifier*** BALB/c susceptibility and ***modifier*** Journal; Article; (JOURNAL ARTICLE) Journal code: NGY, ISSN: 0270-7306. L13 ANSWER 2 OF 13 MEDLINE T1 Efficiency alleles of the Pctr1 AN 2001089278 MEDLINE DN 20565764 plasmacytoma susceptibility. plasmacytomas is a United States polymorphic with included Cdkn2a, sequences for the Taddesse-Heath resistant to CS7BL/6 (1)310-8Cancer СY Д ţ between genes are important in determining phenotype in plant and by genome scanning using recombinant ***congenic*** strains ***loci*** have interactions between ***modifier** ***loci*** have been (R. Fijneman et al., Nat. Genet., 14: 465-467, 1996; T. van Wezzel Nat. Genet., 14: 468-470, 1996; W. N. Frankel et al., Nat. Genet., these quantitative trait loci and their interactions, in particular the *** backcrosses *** using inbred spretus strains (SEG/Pas and on chromosome 7 (Skts1) showed a highly significant interaction were detected between loci on chromosomes 4 and 5, and 16 and The development of cancer is influenced both by exposure to AU Nagase, Hiroki; Mao, Jian-Hua; de Koning, John P.; Minami, hierarchical whole genome scanning of a complete interspecific identified from studies of mouse models of human cancer, and on chromosome 12 (P < 10-16), whereas additional significant L13 ANSWER I OF 13 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE I in interspecific (spretus/musculus) ***backcross*** mice. between skin tumor ***modifier*** ***loci*** can be Epistatic interactions between skin tumor ***modifier*** CS (1) University of California-San Francisco Comprehensive 2340 Sutter Street, San Francisco, CA, 94143 USA SO Cancer Research (February 15, 2001) Vol. 61, No. 4, pp. ***backcross*** (outbred Mus spretus X Mus musculus Skts1-Skts5 interaction, were confirmed in two completely carcinogens and by the host genetic background. Epistatic 371-373, 1996). We demonstrate here that strong genetic systems and are likely to be major contributors to cancer in humans. Several tumor ***modifier*** AN, 2001:151886 BIOSIS PREV200100151886 ISSN: 0008-5472. (NIH/Ola)). A locus Tomoe; Balmain, 1305-1308, print Cancer Center, environmental susceptibility Article English Allan (1) English 15. Some of ***loci*** interactions interactions detected by with Skts5 interactions detected anima 占

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pristane-induced plasma cell tumors over a shorter latency period
products of the Cdkn2a gene have been eliminated, developed
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traditionally susceptible BALB/cAn strain. Biological assays of the p16(INK4a) and p19(ARF) alleles from BALB/c and DBA/2

BALB/c p16(INK4a) allele was less active than its DBA/2

inducing growth arrest of mouse plasmacytoma cell lines and

ras-induced transformation of NIH 3T3 cells, while the two preventing

alleles displayed similar potencies in both assays. We propose that

BALB/c susceptibility/ ***modifier*** ***locus***, Pctrl, is

"efficiency" allele of the p16(INK4a) gene.

Il Method for identifying mutant alleles of mouse affecting a genetic L13 ANSWER 3 OF 13 CAPLUS COPYRIGHT 2001 ACS AN 2000:68594 CAPLUS

locus and their use in screening for human homologs IN Dove, William F.; Shedlovsky, Alexandra

PA Wisconsin Alumni Research Foundation, USA SO PCT Int. Appl., 37 pp.

LA English DT Patent

CODEN: PIXXD2

FAN.CNT 1

APPLICATION NO. KIND DATE PATENT NO.

WO 1999-US15661 PI WO 2000004186 A1 20000127 19990712

DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, II., W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ

JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK,

TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ,

RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, RU, TI, TM

ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CH, CY, DE, DK, CF, CG,

AU 1999-49843 19990712 CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG AU 9949843 A1 20000207 PRAI US 1998-114973 19980714 WO 1999-USI 5661 19990712 AU 9949843

AB A method for breeding mutagenized mice that permits detection

of genetic	LA English	for autoimmune hemolytic anemia. Although these autoimmune
mutagenized founder strain and a second strain of mice carrying an	F3 Priority Journals EM 200102	traits are inherited as a dominant Cabina assessmin in El Librial - Chitta
allele	AB Transgenic mice carrying the RET oncogene under the control of	and most
at a locus that confers the index phenotype. In the test generation,	the	non-New Zealand strains is suppressed due to the contribution of
clusters of individuals are obso. To deviate from the typical phenotype.	metallothionein promoter exhibit severe pigmentation of the whole	wild-type
The genetic material and mols. encoded thereby can be obtained	skiil aliu melanocytic tumors. The genetic background influences melanoma	modifying genes present in the latter strains. Using chromosomal microsatellite markers in the (CSTRL)6, times NYRNEL times
nsing	development	NZB
available methods. Improved and compact methods called ICMM (index-directed cluster-enhanced ***Modifier***	in RET mice: founder mice crossed with BALB/c mice show	***backcross*** progeny, the authors mapped C57BL/6
("incora managed of the state o	ucul cased incidence and increased latency of melanocytic trimors, whereas	AEA produced and an endeal control of the control o
Molecule identification method) are also disclosed. The method is	progeny of	down-regulated by a
exemplified by identification of the suppressor or enhancer alleles	C57BL/6 mice show the opposite effect. Using partially	combined effect of two major independently segregating dominant
or mouse Min allele of APC locus by phenotypic and genotypic	***congenic*** RET mice on a CSTR1/6 censein backerround (N3/RET mice)	alleles,
studies of F1	studied genetic	One littled to D/MH is 90 on chromosome 7 and the other linked to D10MIT42 on
generation (and their outcross and ***backcross*** offsprings)	linkage in (N3/RETxBALB/c)xN3/RET ***backcross*** mice.	chromosome 10. Splenomegaly was modified mainly by a single
ethylnitrosourea mutagenized female BTBR and heterozygous	three melanoma ***modifier*** ***loci***, on chromosome	C3/BL/6 allele linked to D4MIT58 on chromosome 4 Thus the
B6-APCmin/+	l (Melm1	autoimmune hemolytic
male. The identification of these new genes in the mouse disease models	and Melm2) and chromosome 11 (Melm3), that are linked with	anemia in the NZB strain is under multigenic control and a
for human colon cancers are helpful to sercen human homologs	early metanoma incidence and latency. Manning of Melm losi and of five additional	combined action
involved in	regions	of the out susceptibility but also modifying alleres with
the related diseases.	on chromosomes 6, 8, 9, 12, and 13 indicated allelic imbalance in	activities affects the outcome of disease features in the proceny.
KE.CNT 4	N3/RET	There
(1) Anon: GENETICS 1996 V144(4) P1777	mice, with a significant excess of BALB/c alleles, suggesting the	are potentially important candidate genes which may be linked to
(2) Dietrich, W. "GENETIC IDENTIFICATION OF MOM-1, A	of additional putative melanoma ***modifier*** ***!oxi***	wear lation of AEA and advanced.
MAJOR MODIFIER LOCUS		regulation of ALA and spienomegaly. RE.CNT 31
AFFECTING MIN-INDUCED INTESTINAL NEOPLASIA IN	chromosomes.	RE
CAPLUS	113 ANSINED COE 13 CABITIE CONVENTE TO SO	(4) Dietrich, W. Genetics 1992, V131, P423 CAPLUS
(3) Gould, K; Genetic evaluation of candidate genes for the Momi	AN 2000:80654 CAPLUS	(3) Drake, C; Proc Natl Acad Sci USA 1994, V91, P4062 CAPLUS
modifier of		(9) Hirose, S. Int Immunol 1994, V6. P1857 CAPLUS
Intestinal neoplasia in mice	II Genetic regulation of anti-erythrocyte autoantibodies and	(12) Jiang, Y; J Immunol 1997, V158, P992 CAPLUS
(4) Wisconsiii Alailiiii Acs Fourid, WO 9622022 A 1998 CAPLUS	splenomegaly in	ALL CITATIONS AVAILABLE IN THE RE FORMAT
L13 ANSWER 4 OF 13 MEDLINE DUPLICATE	autoinnume nemoiytic aremia-prone New Zealand Black mice AU Ochiai, Kimiko; Ozaki, Shoichi; Tanino, Akihiro; Watanabe,	L13 ANSWER 6 OF 13 MEDLINE
3 AN 2001101647 AGENTARE	Shinji; Ueno,	
	I omoo; Mitsui, Kenichi; Toei, Junichi; Inada, Yuji; Hirose,	AN 2000079602 MEDLINE
Ti Mapping of melanoma ***modifier*** ***loci*** in RET	Shirai, Toshikazu; Nishimura, Hiroyuki	
transgenic	CS Toin Human Science and Technology Center, Department of	
mice. All Emonni T.A. Deiscel B. Zanesi M. Aloisi A. Dai V. Kato M.	Biomedical	survival time of tumor-bearing mice.
Suzuki H;	Engineering, Toin University of Yokohama, Yokohama, 225-8502, Japan	AU Nagase H; Mao J H; Balmain A
Nakashima I	SO Int. Immunol. (2000), 12(1), 1-8	Research
CS Department of Experimental Oncology, Istituto Nazionale Trimori Via G	CODEN: INIMEN; ISSN: 0953-8178	Institute, University of California, 2340 Sutter Street, San
Venezian Milan, Italy. dragani@istitutotumori.mi.it	PB Oxford University Press DT Journal	Francisco, CA 94105 1154
SO JAPANESE JOURNAL OF CANCER RESEARCH, (2000	LA English	SO PROCEEDINGS OF THE NATIONAL ACADEMY OF
Nov) 91 (11) 1142-7. Iournal code: HBA 188N: (010 5050	AB New Zealand Black (NZB) mice spontaneously produce	SCIENCES OF THE UNITED STATES OF
CY Japan	anti-etyturocyte autoantibodies (AEA) in assocn, with snlenomegaly, thus serving as	AMERICA, (1999 Dec 21) 96 (26) 15032-7.
DT Journal; Article; (JOURNAL ARTICLE)	a model	Journal code: PV3, ISSN: U027-8424. CY United States

DT Journal: Article: (JOURNAL ARTICLE)	SO PROCEEDINGS OF THE NATIONAL ACADEMY OF	SADA STATES
-	SCIENCES OF THE UNITED STATES OF	OADA-etgic pathways.
For Priority Journals; Cancer Journals EM 200003	AMERICA, (1997 Dec 9) 94 (25) 14060-5. Journal code: DV3 1SSN: 0027-8424	THE ANSITED 9 OF 12 MENTINE
EW 20000305	CY United States	LIS ANSWERS OF IS INELLINE 6
AB Studies of mouse models of human cancer have established the	DT Journal; Article; (JOURNAL ARTICLE)	AN 96172827 MEDLINE
existence of		DN 96172827
multiple tumor modifiers that influence parameters of cancer		TI Modulation of disease severity in cystic fibrosis transmembrane
susceptionity such as turnor multiplicity, turnor size, or the probability	EM 199803	conductance regulator deficient mice by a secondary genetic factor
of malignant progression. We have carried out an analysis of skin		[published erratum appears in Nat Genet 1996 May, 13(1):129].
tumor		Aughbach W. Moore
susceptibility in interspecific Mus musculus/Mus spretus hybrid	the mammalian brain, is synthesized by two glutamate	A: Forstner J: Durie P: Nadeau J: Bear C: Tsui I. C
mice and	decarboxylase	CS Department of Molecular Genetics, The University of Toronto.
have identified another seven loci showing either significant (six	isoforms, GAD65 and GAD67. The separate role of the two	Ontario,
[00]	isoforms is	Canada.
or suggestive (one locus) linkage to tumor susceptibility or	unknown, but differences in saturation with cofactor and subcellular	SO NATURE GENETICS, (1996 Mar) 12 (3) 280-7.
resistance, A	localization suggest that GAD65 may provide reserve pools of	Journal code: BRO. ISSN: 1061-4036.
specific search was carried out for skin tumor "modifier"	GABA for	CY United States
associated with time of survival after development	regulation of inhibitory neurotransmission. We have disrupted the	DT Journal, Article; (JOURNAL ARTICLE)
	gene	LA English
inalignali tambi. A combination of resistance affers at ince- mariere	encoding GALNO3 and """backcrossed""" the mutation into the	FS Priority Journals
[D6Mit15 (Skre12) D7Mit12 (Skrs2) and D17Mit15 (Skrs10)1 all	C3/BL/0	EM 199605
of which	stain of miles. In compast to CALCO1-1- animals, which are born units	AB Mice that have been made delicient for the cystic librosis
are close to or the same as loci associated with carcinoma incidence	Will) derestonmental abnormalities and dis about 1. offer bish CADCE !	transmembrane
and/or nanilloma multiplicity is significantly associated with	mice	conductance regulator (Citr) usually die of intestinal obstruction.
increased	annear normal at high Basal GABA layels and holy GAD activity.	Less series of the series of t
survival of mice with carcinomas, whereas the reverse combination	are	nave created City-delicient mice and demonstrate protonged
Jo	normal, but the pyridoxal 5' phosphate-inducible ano-enzyme	survival annuig
susceptibility alleles is significantly linked to early mortality caused	reservoir is	Strains.
by rapid carcinoma growth (chi(2) = 25.22; $P = 5.1 \times 10(-8)$). These	significantly decreased. GAD65-/- mice develop spontaneous	suggesting that modulation of disease severity is penetically
data	seizures that	determined.
indicate that host genetic factors may be used to predict carcinoma	result in increased mortality. Seizures can be precipitated by fear or	A genome scan showed that the major ***modiffer***
growth	mild stress. Seizure susceptibility is dramatically increased in	***locus***
rate and/or survival of individual ***backcross*** muce exposed	GAD65-/-	maps near the centromere of mouse chromosome 7.
to the	mice ***backcrossed*** into a second genetic background, the	Electrophysiological
same carcinogenic stimulus and suggest that mouse models may	nonobese	studies on mice with prolonged survival show that the partial
provide an anarcach to the identification of cenatic modifiers of cenasis	diabetic (NOLD/LIJ) strain of mice enabling electroencephalogram	rectification of CI- and Na+ ion transport abnormalities can be
survival in	diddysis of the coiming The consorbit higher hand benin GADA Janua in	explained
humans.	this	in part by up-regulation of a calcium-activated Ci-conductance. Identification of modifier sense in our Office UNSONORY ILISON
	backcross are significantly decreased by the GAD65-/-	
L13 ANSWER 7 OF 13 MEDLINE DUPLICATE	mutation,	should provide important insight into the heterogeneous disease
	suggesting that the relative contribution of GABA synthesized by	presentation observed among CF patients.
AN 1998034360 MEDLINE DN 98054360	GAD65 to	
	total brain GALA revels is genetically determined. Seizure-associated	L13 ANSWER 9 OF 13 MEDLINE DUPLICATE 7
decarboxylase.	c-fos-like immunoreactivity reveals the involvement of limbic	AN 96121384 MEDIINE
AU Kash S F; Johnson R S: Tecott L H; Noebels J L; Mayfield R D;	regions of	96121384
Hanahan D,	the brain. These data suggest that GABA synthesized by GAD65 is	_
Backkeskov S	important	
C3. Department of Medicine, School of Medicine, University of	in the dynamic regulation of neural network excitability, implicate	17.
San Francisco San Francisco CA 94143 118A		
NC DK41822 (NIDDK)	strain, and	CS Jackson Laboratory, Bar Harbor, Maine (1460), USA. NC HT28882 (NICHT)
NS29709/11535 (NINDS)	present GAD65-/- animals as a model of epilepsy involving	SO GENOMICS (1995 Oct 10) 29 (3) 719,24

AB Mutations in the human APC gene caused various familial colon gene and develops many intestinal adenomas. Here, we analyze DT Journal; Article; (JOURNAL ARTICLE) FS Priority Journals; Cancer Journals frequent somatic conversion of steroid sulfates to their active nonconjugated form. In activity was measured in the ***backcross*** strains SHR/y and hypertension. An alternative hypotheses is that a regulatory locus in addition to the structural locus is responsible for STS activity levels in these strains were intermediate between those of SHR and mammals the steroid sulfatase locus (Sts) is on the Y chromosome, and this regulatory locus is on the rat Y chromosome. Further study hypothesis can be eliminated, the Sis locus or its ***modifier*** DUPLICATE ***loci*** remain a potential component of the Y chromosome STS activity in testes, adrenal gland, liver, and hypothalamus. The values for STS in the two strains were not significantly different; AU Dietrich W F; Lander E S; Smith J S; Moser A R; Gould K A; needed to discriminate between these possibilities, and until the between SHR and WKY, the STS activity could be a secondary the rat Sts is on the X chromosome. We measured STS activity to test and/or confirm a Y chromosome influence on STS. STS CS Whitehead Institute for Biomedical Research, Massachusetts activity differences were likely due to differences in enzyme TI Genetic identification of Mom-1, a major ***modifier*** Because the blood pressures of SHR/y and SHR/a were also and normotensive Wistar Kyoto (WKY) males. SHR had affecting Min-induced intestinal neoplasia in the mouse Journal; Article; (JOURNAL ARTICLE) CELL, (1993 Nov 19) 75 (4) 631-9. Journal code: CQ4, ISSN: 0092-8674. L13 ANSWER 11 OF 13 MEDLINE Technology, Cambridge 02142. NC HG00098 (NHGRI) AN 94061981 MEDLINE Borenstein N. Dove W. HG00126 (NHGRJ) CA07075 (NCI) United States response to the 861907 amounts. STS hypertensive activity CY S and Mus spretus. No effect of met I was seen in a higher penetrance T1 Steroid sulfatase and the Y chromosome hypertensive locus of the Midwest Hypertension Research Center, Omaha, Nebraska, USA. STEROIDS, (1995 Oct) 60 (10) 681-5.
Journal code: V10. ISSN: 0039-128X. overall. No evidence was found for epistatic interaction between ct all of the affected individuals in the BALB/cByJ cross and most of DUPLICATE The spontaneously hypertensive rat (SFIR) has a Y chromosome AB The major gene for neural tube defects, ct, in the curly-tail (CT) modifiers or strain-specific susceptibility alleles. Here we describe increases blood pressure. This locus requires an androgen receptor mapping of a curly-tail ***modifier*** ***locus***, mctl, with the BXD-8/Ty strain, confirming that ct is the major gene in affected individuals in the M. spretus cross and was the preferred trait, already incomplete in the CT strain, was further reduced in data from several ***backcrosses*** . The penetrance of the model. Homozygosity at both ct and mct l loci was sufficient to chromosome 17 in moderate and low penetrance crosses of CT testosterone for maximum expression. Steroid sulfatase (STS) of these ***backcrosses*** , suggesting the existence of strain was mapped previously to mouse chromosome 4 by Journal; Article; (JOURNAL ARTICLE) Journal; Article; (JOURNAL ARTICLE) AU Johnson M.L. Ely D.L., Turner M.E. CS Midwest Hypertension Research Cen SO STEROIDS, (1995 Oct.) 60 (10) 681. Journal code: GEN, ISSN; 0888-7543. L13 ANSWER 10 OF 13 MEDLINE spontaneously hypertensive rat. AN 96106991 MEDLINE Priority Journals Priority Journals United States United States combining linkage with BALB/cByJ 6690196 EM 199604 English English catalyzes the neural tub locus that E S E CY Ω FS E the the th

- syndromes. The Multiple intestinal neoplasia (Min) mouse
- excellent model for familial colon cancer: it carries a mutant mouse
- tumor phenotype is dramatically modified by genetic background.
- the genetic mapping of a locus that strongly modifies turnor number Min/+ animals. This gene, Mom-I (Modifier of Min-I), maps to
- chromosome 4 and controls about 50% of genetic variation in tumor
 - in two intraspecific ***backcrosses*** . The mapping is supported by a
- LOD score exceeding 14. Interestingly, Mom-1 lies in a region of conservation with human chromosome 1p35-36, a region of
- loss of heterozygosity in a variety of human tumors, including colon tumors. These results provide evidence of a major modifier
- expression of an inherited cancer syndrome
- DUPLICATE L13 ANSWER 12 OF 13 MEDILINE
- AN 92176249 MEDLINE DN 92176249
- Tl The Min (multiple intestinal neoplasia) mutation: its effect on gut epithelial cell differentiation and interaction with a modifier
- AU Moser A R; Dove W F; Roth K A; Gordon J I CS McArdle Laboratory, University of Wisconsin, Madison 53706.
 - NC CA07075 (NCI)
- CA50585 (NCI) CA23076 (NCI)
- SO JOURNAL OF CELL BIOLOGY, (1992 Mar) 116 (6) 1517-26. Journal code: HMV, ISSN: 0021-9525.
 - CY United States
- FS Priority Journals; Cancer Journals
- AB Min is a fully penetrant dominant mutation that leads to the
- of multiple intestinal adenomas throughout the duodenal-to-colonic
- Min/+ C57BL6/J mice have an average life-span of 120 d.
- immunocytochemical studies of these lesions demonstrate patches

populations for selection procedures that exploit specific combining ability efects. Together with higher frequencies of BCI times. BCI testerosses superior to B73. times. Mol 7H, they indicated a higher ntrogression of broadbase germplasm to improve the elite single of the modifier allele in the two composites would be near zero which causes complete dominance at a quantitative trait locus. hybrids is an important objective of maize breeding programs. model. The VarSCA results suggested the use of => e dove william f/au Mol 7H(Mol 7H ***backcross*** interpopulation interpopulation Proportion of (VarSCA) for and Mol7H two-allele Frequency a [B73 73 cross B73 Design 2 and four E1 E2 E3 E4 E5 E5 E6 E7 Preliminary *** backcross *** analysis is consistent with a single adenomas appear to be established by 100 d of age or sooner. These fully penetrant in hybrids with either AKR/I or MA/MyJ. However, Paneth cells. Expression of endogenous marker genes within these by the adenoma along the duodenal-to-colonic axis and mirrors the were sought in which Min/+ animals could survive for up to 300 d. differentiation in the continuously renewing gut epithelium and for AU BERNARDO R; JOHNSON G R; DUDLEY J W; MEGHJI M associated with the development of invasive tumors. New tumors To study the time-dependent properties of these tumors, genetic MA/MyJ strains. The increased lifespan of the hybrid animals is differentiated enterocytes, and scattered enteroendocrine, goblet arise continuously over the lifespan of these animals; instead all suggests that tumorigenesis in Min/+ mice may be initiated in a ***modifier*** ***locus*** unlinked to Min in both the differentiated cells can be directly correlated with the position multipotent stem cell normally located at the base of intestinal AB Further improvement in the performance of elite maize (Zca assessment of the multi-step hypothesis of intestinal neoplasia. L13 ANSWER 13 OF 13 BIOSIS COPYRIGHT 2001 BIOSIS differentiation of the normal gut epithelium. The presence of indicate that the Min/+ mouse is a powerful model system for EVALUATION OF F-2 X F-2 AND BC-1 X BC-1 MAIZE ineages in adenomas together with their retention of spatial CS DEP. AGRON., UNIV. ILL., 1102 S. GOODWIN AVE., URBANA, ILL. 61801. hybrids demonstrate a reduction in the number of intestinal mechanisms that establish and maintain a balance between CODEN: CRPSAY, ISSN: 0011-183X SO CROP SCI, (1989) 29 (6), 1377-1381 INTERPOPULATION CROSSES. 1990:43589 BIOSIS BA89:20953 proliferation and FS BA; OLD LA English analyzing the AKR/J and conditions multiple mays L.) ΑN ΣΩ 뛽

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TI Method for identifying mutant alleles of mouse affecting a genetic
                                                                                                                                                                                                                              133 ("DOVE WILLIAM"/AU OR "DOVE WILLIAM
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  7 DUP REM LIS (5 DUPLICATES REMOVED)
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  IN ***Dove, William F.***; Shedlovsky, Alexandra
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                locus and their use in screening for human homologs
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             PA Wisconsin Alumni Research Foundation, USA
DOVECAR FRANK/AU
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                                                                                                                                                                                                                                                                                                                                                                        broadbase gemplasm had a linear effect on means with the order of performance (most favorable to least favorable) for all traits being
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 genetic model. Contrary to expectations, estimates for grain yield of CovHS in the population related to B73 and of VarSCA were two
                                                                   times. Mol 7H was considered. Estimates of genetic parameters in
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              cross, and A Composite .times. B Composite. Estimates of half-sib
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    grain moisture and plant and ear heights were greater in the F2 than
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          ***locus***
                                                                                                                                             times. B Composite]F2 .times. [Mo17H .times. A Composite]F2
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                            cross were consistent with expectations for a model in which B73
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                times greater, respectively, in the BC1 than in the F2. The large estimates of CovHS and VarSCA for grain yield in the BC,
                                                                                                                                                                                                                                                                                          times. A Composite)]BC1 Design 2 population were obtained.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   covariances (CovHS) and specific combining ability variance
                                                                                                                                                                                                                       population and a [B73(B73 .times. B Composite]BC1 .times.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    the BC1 interpopulation cross as expected for a one-locus,
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       times. Mol 7H, the BC1 interpopulation cross, the F2
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          are homozygous for an allele at a ***modifier***
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DOVE YVONNE/AU

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W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH,
                                                                                                       DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, II.
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           WO 1999-USI 5661
      PI WO 2000004186 A1 20000127
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probability for immediate derivation of superior single crosses from
                                                                                                                                                                                                                                                 DOVE WILLIAM GAU
DOVE WILLIAM TAU
DOVE WILLIAM THOMASON/AU
DOVE WINIFRED/AU
DOVE Y/AU
                                                                                                                                                                                DOVE WAYNE KEITH/AU
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APPLICATION NO.

KIND DATE

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DT Patent LA English

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SO PCT Int. Appl., 37 pp. CODEN: PIXXD2

ES, FI, FR, GB, GR, IE, IT, LU, MC, NI,, PT, SE, BF, BJ

CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG 9949843 A1 20000207 AU 1999-49843 19990712 PRAI US 1998-114973 19980714

AB A method for breeding mutagenized mice that permits detection WO 1999-US15661 19990712

loci that can modify a known index phenotype involves crossing a mutagenized founder strain and a second strain of mice carrying an

at a locus that confers the index phenotype. In the test generation, clusters of individuals are obsd. to deviate from the typical

The genetic material and mols, encoded thereby can be obtained

available methods. Improved and compact methods called ICMM (index-directed, cluster-enhanced, ***Modifier***

Molecule identification method) are also disclosed. The method is

mouse Min allele of APC locus by phenotypic and genotypic studies of F1

exemplified by identification of the suppressor or enhancer alleles

ethylnitrosourea mutagenized female BTBR and heterozygous generation (and their outcross and backcross offsprings) of B6-APCmin male. The identification of these new genes in the mouse disease

for human colon cancers are helpful to screen human homologs involved in

the related diseases.

RE.CNT 4

(1) Anon; GENETICS 1996, V144(4), PI 777 (2) Dietrich, W; "GENETIC IDENTIFICATION OF MOM-1, A MAJOR MODIFIER LOCUS

AFFECTING MIN-INDUCED INTESTINAL NEOPLASIA IN THE MOUSE" CELL V75, P631

(3) Gould, K; Genetic evaluation of candidate gencs for the Moml

(4) Wisconsin Alumni Res Found; WO 9822622 A 1998 CAPLUS

intestinal neoplasia in mice

L16 ANSWER 2 OF 7 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE

AN 2000:343967 BIOSIS

DN PREV200000343967

The MornI AKR intestinal turnor resistance region consists of

locus distal to D4Mit64.

AU Cormier, Robert T.; Bilger, Andrea; Lillich, Amy J.; Halberg, Hong, Karen H.; Gould, Karen A.; Borenstein, Natalie; Lander,

***Dove, William F. (1)**

CS (1) McArdle Laboratory for Cancer Research, University of

SO Oncogene, (29 June, 2000) Vol. 19, No. 28, pp. 3182-3192. print. ISSN: 0950-9232. Madison, WI, 53706 USA

DT Article

LA English SL English

AB The Mom1 (Modifier of Min-1) region of distal chromosome 4 was identified

during a screen for polymorphic modifiers of intestinal tumorigenesis in ApcMin/+ mice. Here, we demonstrate that the Mom1AKR allele

two genetic components. These include the secretory phospholipase consists of

whose candidacy as a Mom1 resistance modifier has now been tested with several transgenic lines. A second region, distal to Pla2g2a, has also been identified using fine structure recombinants. Pla2g2aAKR transgenic mice demonstrate a modest resistance to tumorigenesis in the small intestine and a very robust resistance in the large intestine.

Moreover.

dosage-dependent, a finding that is consistent with our observation the tumor resistance in the colon of Pla2g2aAKR animals is

Pla2g2a is expressed in goblet cells. By contrast, mice carrying the distal Mom1 modifier demonstrate a modest tumor resistance that

modifier ***loci*** are complementary, both in their quantitative and regional effects. The additive effects and tight confined to the small intestine. Thus, the phenotypes of these two

of these modifiers may have been necessary for the initial identification

of the Mom1 region

Il The intestinal epithelium and its neoplasms: Genetic, cellular and L16 ANSWER 3 OF 7 BIOSIS COPYRIGHT 2001 BIOSIS AN 1998:394514 BIOSIS DN PREV199800394514

interactions

Dove, William F.; Cormier, Robert T.; Gould, Karen Richard B.; Merritt, Anita J.; Newton, Michael A.; Shoemaker,

Alexander R.

CS McArdle Lab. Cancer Res., Univ. Wisconsin, Madison, WI 53706 USA

SO Philosophical Transactions of the Royal Society of London B

Sciences, (June 29, 1998) Vol. 353, No. 1370, pp. 915-923. ISSN: 0962-8436.

General Review

AB The Min (multiple intestinal neoplasia) strain of the laboratory

its derivatives permit the fundamental study of factors that regulate

importance in mediating these alternative patterns of growth is Apc, transition between normal and neoplastic growth. A gene of central

mouse homologue of the human adenomatous polyposis coli (APC)

inactivated. One copy is mutated by the nonsense Apc allele carried adenomas form in the Min mouse, both copies of the Ape gene

heterozygous form in this strain. The other copy can be silenced by

several mechanisms. These range from loss of the homologue wild-type Apc allele; to interstitial deletions surrounding the bearing the

allele; to intragenic mutation, including nonsense alleles; and wild type

to a reduction in expression of the locus, perhaps owing to mutation

regulatory locus. Each of these proposed mechanisms may

two-hit genetic process as initially posited by Knudson; however, apparently the two hits could involve either a single locus or two The kinetic order for the transition to adenoma may be still higher two, if polyclonal adenomas require stronger interactions than

fusion. The severity of the intestinal neoplastic phenotype of the

mouse is strongly dependent upon loci other than Apc. One of has now been rigorously identified at the molecular level as these, Moml.

active resistance conferred by a secretory phospholipase. Mom1 encoding an

lineage, however, its action seems to be non-autonomous: when locally within a crypt lineage, not systemically. Within the crypt

arise in Mom1 heterozygotes, the active resistance allele is maintained in the tumour (MOH or maintenance of heterozygosity). Indeed, the phospholipase is synthesized by post-mitotic Paneth cells, not by proliferative cells that presumably generate the tumour. An analysis

autonomy of modifier gene action in chimeric mice deserves

chimeric and to the clonal structure of the tissue in question.

attention both to the number of genetic factors for which an animal

Mom1, other loci can strongly modify the severity of the Min

multiplicity in Min mice. Recent evidence suggests that Mom1 may on tumor development. However, within the colonic grafts, the Min mapped one of these ***modifier** ***loci***, Moml, to secretory phospholipase, Pla2g2a. Pla2g2a is expressed in a variety cell types and seems to be involved in inflammatory responses and phenotype does not appear to be autonomous; the development of LA English AB Mutations In the human A PC gene cause various familial colon throughout the intestinal tract. Tumor multiplicity in Min mice is influenced by genetic ***modifier*** ***(loci*** mouse chromosome 4. Morn1 is a semidominant modifier of both AU Dietrich, William F. (1); Lander, Eric S. (1); Smith, Jennifer S. secretions, dietary components, or intestinal flora, may be critical bacterial defense mechanisms. Here, we determine whether Min tumors in Min mice seems dependent on factors beyond the Min in a tissue-autonomous fashion using ectopic intestinal isografts. factors contributing to the development of Min-induced colonic However, these factors are not required for the action of Min or Moser, Amy R.; Gould, Karen A.; Luongo, Cindy; Borenstein, manner. There is no evidence that either Min or Mom1 has a TI Genetic identification of Mom-1, a major ***modifier*** the colonic epithelium. Microenvironmental factors, such as L16 ANSWER 7 OF 7 BIOSIS COPYRIGHT 2001 BIOSIS CS (1) Whitehead Inst. Biomed Res., Dep. Biol., Mass. Inst. syndromes. The Multiple intestinal neoplasia (Min) mouse the small intestinal grafts, both Min and Mom1 act in a affecting Min-induced intestinal neoplasia in the mouse. SO Cell, (1993) Vol. 75, No. 4, pp. 631-639. ISSN: 0092-8674. Cambridge, MA 02142 USA within the small intestine ***Dove, William*** AN 1994:64047 BIOSIS DN PREV199497077047 lissue-autonomous **DUPLICATE 5** systemic effect tumor size and and Mom l act ***locus DT Article Previously, digestive Natalie; account for such modifications. The Min mouse strain, in which the ***modifier*** ***locus*** , Mom1 (modifier of Min-1), to TI Action of Min and Mom1 on neoplasia in ectopic intestinal grafts.

AU Gould, Karen A.; ***Dove, William F. (1)***
CS (1) McArdle Lab. Cancer Research, 1400 University Ave., gene, develops intestinal neoplasms whose multiplicity is strongly a secretory phospholipase. Here, we report that a cosmid transgene Pla2g2a with Mom1 thus withstands a strong functional test and is tool to identify factors in the environment and genetic background CS (1) McArdle Lab. Cancer Research, Univ. Wisconsin, Madison, AB Individuals inheriting the same mutation predisposing to cancer overexpressing Pla2g2a caused a reduction in tumour multiplicity disease-free survival. Experimental mouse models can provide a very different outcomes, ranging from early aggressive cancer to affected by genetic background. We previously mapped a strong mutation disrupts the mouse homologue of the human familial comparable to that conferred by a single copy of the resistance AB Mice heterozygous for Min, a mutant allele of Apc, develop L.; Richardson, Paul; Mulherkar, Rita, ***Dove, William F. Mom1. These results offer strong evidence that this secretory phospholipase can provide active tumour resistance. The region on mouse chromosome 4 containing a candidate gene L16 ANSWER 6 OF 7 BIOSIS COPYRIGHT 2001 BIOSIS SO Cell Growth & Differentiation, (1996) Vol. 7, No. 10, pp. to represent the successful identification of a polymorphic SO Nature Genetics, (1997) Vol. 17, No. 1, pp. 88-91 AN 1996:527044 BIOSIS DN PREV199699249400 trait locus in mammals. ISSN: 1061-4036. Madison, WI 53706 ISSN: 1044-9523. Pla2g2a encoding Lander, Eric S. **DUPLICATE 4** Hawkins, Trevor WI 53706 USA LA English DT Article English DT Article quantitative 1361-1368. adenomas may show and size, a 4-cM migration, adhesion, and polarity. Adenoma multiplicity and growth secretory phospholipase Pla2g2a is a candidate for Mom1. Here, we mice indicates that the actions of both Apc and Mom1 are localized human families, one can investigate by a candidate approach which interact with the intestinal cancer predisposition of the Min mouse AU Gould, Karen A.; ***Dove, William F. (1)*** CS (1) McArdle Lab. Cancer Res., 1400 University Ave., Madison, WI 53706 USA SO Proceedings of the National Academy of Sciences of the United emergent challenge is to find ways to identify the full set of genes Secretory phospholipase Pla2g2a confers resistance to intestinal are modulated by an unlinked ***modifier*** ***locus*** throughout the intestinal tract. Apc is believed to be involved in AB Mice heterozygous for the Apc-Min (Min) mutation develop mouse genetics provide candidates for chemopreventive and/or Tl Localized gene action controlling intestinal neoplasia in mice. strain. With such a set, one can then work, using contemporary investigate the range of action of Apc and Mom1. Analysis of modifying factors influence the epidemiology of human colon L16 ANSWER 5 OF 7 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 3 L16 ANSWER 4 OF 7 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 2 AU Cormier, Robert T.; Hong, Karen H.; Halberg, Richard B.; genetics, to identify the molecular, cellular and organismal if a candidate modifier does not explain any of the genetic of colon cancer in human populations, modifier activities that integrate their functions. Finally, with appropriately the cell lineage that gives rise to intestinal tumors. America, (1997) Vol. 94, No. 11, pp. 5848-5853. 1997:438664 BIOSIS AN 1997:298155 BIOSIS modalities in the human. PREV19979737867 PREV199799597358 ISSN: 0027-8424. phenotype. An discovered by chimeric Min epidemiology cancer. Even DT Article LA English Moml. The phenotyped adenomas States of Z 믕 rate Z

excellent model for familial colon cancer; it carries a mutant mouse

gene and develops many intestinal adenomas. Here, we analyze

tumor phenotype is dramatically modified by genetic background,

the genetic mapping of a locus that strongly modifies tumor number

Minl+ animals, This gene, Mom-1 (Modifier of Min-1), maps to

chromosome 4 and controls about 50% of genetic variation In

in two by a LOD score exceeding 14. Interestingly, Mom-1

backcrosses. The mapping is supported by a LOD score exceeding

Interesting Mom-1 lies in a region of synteny conservation with

chromsome 1p35-36, a region of frequent somatic loss of

a variety of human tumors, including colon tumors. These results heterozygosity

evidence of a major modifier affecting expression of an inherited

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SHEDLOVSKY JULIAN PYAU

SHEDLOVSKY LEO/AU

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L21 ANSWER 1 OF 7 CAPLUS COPYRIGHT 2001 ACS AN 2000:68594 CAPLUS DN 132:103741

TI Method for identifying mutant alleles of mouse affecting a genetic

locus and their use in screening for human homologs

Shedlovsky, Alexandra ***Dove, William F. *** Z

PA Wisconsin Alumni Research Foundation, USA SO PCT Int. Appl., 37 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT

APPLICATION NO. KIND DATE PATENT NO. WO 1999-US15661 PI WO 2000004186 AI 20000127 W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, C DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD

MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ MG, MK

TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY RU, TJ, TM KG, KZ, MD

RW: GH, GM, KE, LS. MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL. PT, SE, BF, BJ

AU 1999-49843 19990712 CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG 9949843 A1 20000207 AU 1999-49843 I PRAI US 1998-114973 19980714 AU 9949843

WO 1999-US15661 19990712

AB A method for breeding mutagenized mice that permits detection

mutagenized founder strain and a second strain of mice carrying an loci that can modify a known index phenotype involves crossing a

at a locus that confers the index phenotype. In the test generation, clusters of individuals are obsd. to deviate from the typical

The genetic material and mols, encoded thereby can be obtained

available methods. Improved and compact methods called ICMM (index-directed, cluster-enhanced, ***Modifier***

locus and

Molecule identification method) are also disclosed. The method is exemplified by identification of the suppressor or enhancer alleles

mouse Min allele of APC locus by phenotypic and genotypic

ethylnitrosourea mutagenized female BTBR and heterozygous generation (and their outcross and backcross of springs) of B6-APCmin/+

male. The identification of these new genes in the mouse disease

for human colon cancers are helpful to screen human homologs involved in

the related diseases.

RE.CNT 4

(1) Anon; GENETICS 1996, V144(4), P1777

(2) Dietrich, W; "GENETIC IDENTIFICATION OF MOM-1, A

MAJOR MODIFIER LOCUS

AFFECTING MIN-INDUCED INTESTINAL NEOPLASIA IN THE MOUSE" CELL V75, P631

(3) Gould, K; Genetic evaluation of candidate genes for the Moml modifier of

intestinal neoplasia in mice

(4) Wisconsin Alumni Res Found; WO 9822622 A 1998 CAPLUS

L21 ANSWER 2 OF 7 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE

AN 2000:343967 BIOSIS DN PREV200000343967

II The Moml AKR intestinal tumor resistance region consists of Pla2g2a and a

locus distal to D4Mit64

AU Cormier, Robert T.; Bilger, Andrea, Lillich, Amy J.; Halberg, Richard B.:

Hong, Karen H.; Gould, Karen A.; Borenstein, Natalie; Lander,

Dove, William F. (1)

CS (1) McArdle Laboratory for Cancer Research, University of Wisconsin,

SO Oncogene, (29 June, 2000) Vol. 19, No. 28, pp. 3182-3192. print ISSN: 0950-9232. Madison, WI, 53706 USA

DT Article

LA English SL English AB The Mom! (Modifier of Min-1) region of distal chromosome 4

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phenotype. An these, Moml, encoding an phenotyped secretory detailed finally. passive Μin the ō .2 several transgenic lines. A second region, distal to Pla2g2a, has also mouse homologue of the human adenomatous polyposis coli (APC) mice demonstrate a modest resistance to tumorigenesis in the small ***modifier*** ***loci*** are complementary, both in their importance in mediating these alternative patterns of growth is Apc, inactivated. One copy is mutated by the nonsense Apc allele carried The intestinal epithelium and its neoplasms: Genetic, cellular and its derivatives permit the fundamental study of factors that regulate transition between normal and neoplastic growth. A gene of central dosage-dependent, a finding that is consistent with our observation Pla2g2a is expressed in goblet cells. By contrast, mice carrying the ApcMin/+ mice. Here, we demonstrate that the Mom1AKR allele distal Mom1 modifier demonstrate a modest tumor resistance that confined to the small intestine. Thus, the phenotypes of these two ***Dove, William F. *** : Cormier, Robert T.: Gould, Karen The Min (multiple intestinal neoplasia) strain of the laboratory been identified using fine structure recombinants. Pla2g2aAKR SO Philosophical Transactions of the Royal Society of London B whose candidacy as a Mom1 resistance modifier has now been Richard B.; Merritt, Anita J.; Newton, Michael A.; Shoemaker, adenomas form in the Min mouse, both copies of the Apc gene quantitative and regional effects. The additive effects and tight CS McArdle Lab. Cancer Res., Univ. Wisconsin, Madison, WI the tumor resistance in the colon of Pla2g2aAKR animals is L21 ANSWER 3 OF 7 BIOSIS COPYRIGHT 2001 BIOSIS Sciences, (June 29, 1998) Vol. 353, No. 1370, pp. 915-923. intestine and a very robust resistance in the large intestine. of these modifiers may have been necessary for the initial two genetic components. These include the secretory 1998:394514 BIOSIS PREV199800394514 of the Moml region. phospholipase Pla2g2a, General Review ISSN: 0962-8436. Alexander R. English A.; Halberg, 53706 USA Biological Ā NO 占 Ŋ

tumorigenesis in

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migration, adhesion, and polarity. Adenoma multiplicity and growth
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AU Gould, Karen A.; ***Dove, William F. (1)***
CS (1) McArdle Lab. Cancer Res., 1400 University Ave., Madison, WI 53706 USA
SO Proceedings of the National Academy of Sciences of the United States of
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AB Individuals inheriting the same mutation predisposing to cancer
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TI Secretory phospholipase Pla2g2a confers resistance to intestinal
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modifying factors influence the epidemiology of human colon
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                                                                                if a candidate modifier does not explain any of the genetic
                                                                                                                                                                   of colon cancer in human populations, modifier activities
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                              heterozygous form in this strain. The other copy can be silenced by
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The kinetic order for the transition to adenoma may be still higher
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          two-hit genetic process as initially posited by Knudson; however, apparently the two hits could involve either a single locus or two
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     the tumour (MOH or maintenance of heterozygosity). Indeed, the
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        emergent challenge is to find ways to identify the full set of genes
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               mouse is strongly dependent upon loci other than Apc. One of
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                                                                                                                                                                                                                                                                                      allele; to intragenic mutation, including nonsense alleles; and
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                            Mom1, other loci can strongly modify the severity of the Min
                                                                                                                                                                                                  wild-type Apc allele; to interstitial deletions surrounding the
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       has now been rigorously identified at the molecular level as
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     arise in Mom1 heterozygotes, the active resistance allele is
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        chimeric and to the clonal structure of the tissue in question.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          genetics, to identify the molecular, cellular and organismal
                                                                                                                                                                                                                                                                                                                                                                                                                                                      regulatory locus. Each of these proposed mechanisms may
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          that integrate their functions. Finally, with appropriately
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on turnor development. However, within the colonic grafts, the Min cell types and seems to be involved in inflammatory responses and bacterial defense mechanisms. Here, we determine whether Min excellent model for familial colon cancer: it carries a mutant mouse the genetic mapping of a locus that strongly modifies turnor number phenotype does not appear to be autonomous; the development of AB Mutations In the human A PC gene cause various familial colon AU Dietrich, William F. (1); Lander, Eric S. (1); Smith, Jennifer S. tumors in Min mice seems dependent on factors beyond the Min secretions, dietary components, or intestinal flora, may be critical tumor phenotype is dramatically modified by genetic background, Minl+ animals. This gene, Mom-1 (Modifier of Min-1), maps to in a tissue-autonomous fashion using ectopic intestinal isografts. factors contributing to the development of Min-induced colonic However, these factors are not required for the action of Min or gene and develops many intestinal adenomas. Here, we analyze Moser, Amy R.; Gould, Karen A.; Luongo, Cindy; Borenstein, manner. There is no evidence that either Min or Mom1 has a chromosome 4 and controls about 50% of genetic variation In Il Genetic identification of Mom-1, a major ***modifier*** the colonic epithelium. Microenvironmental factors, such as L21 ANSWER 7 OF 7 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 5 syndromes. The Multiple intestinal neoplasia (Min) mouse CS (1) Whitehead Inst. Biomed Res., Dep. Biol., Mass. Inst. the small intestinal grafts, both Min and Mom1 act in a affecting Min-induced intestinal neoplasia in the mouse. SO Cell, (1993) Vol. 75, No. 4, pp. 631-639. Cambridge, MA 02142 USA within the small intestine. DN PREV199497077047 ***Dove, William*** AN 1994:64047 BIOSIS ISSN: 0092-8674. issue-autonomous systemic effect and Mom1 act ***!ocus*** LA English genotype of DT Article provides an We report digestive Technol., how this Natalie; colonic Moml distal account for such modifications. The Min mouse strain, in which the ***modifier*** ***locus*** . Mom! (modifier of Min-1), to TI Action of Min and Mom1 on neoplasia in ectopic intestinal grafts. AU Gould, Karen A.: ***Dove, William F. (1)*** a secretory phospholipase. Here, we report that a cosmid transgene secretory phospholipase, Pla2g2a. Pla2g2a is expressed in a variety mapped one of these ***modifier** ***loci***, Moml, to tool to identify factors in the environment and genetic background gene, develops intestinal neoplasms whose multiplicity is strongly Pla2g2a with Mom1 thus withstands a strong functional test and is overexpressing Pla2g2a caused a reduction in turnour multiplicity mouse chromosome 4. Mom1 is a semidominant modifier of both throughout the intestinal tract. Tumor multiplicity in Min mice is influenced by genetic ***modifier*** ***loci**** disease-free survival. Experimental mouse models can provide a very different outcomes, ranging from early aggressive cancer to affected by genetic background. We previously mapped a strong mutation disrupts the mouse homologue of the human familial multiplicity in Min mice. Recent evidence suggests that Mom1 comparable to that conferred by a single copy of the resistance Mom1. These results offer strong evidence that this secretory AB Mice heterozygous for Min, a mutant allele of Apc. develop region on mouse chromosome 4 containing a candidate gene CS (1) McArdle Lab. Cancer Research, 1400 University Ave., Madison, WI 53706 L21 ANSWER 6 OF 7 BIOSIS COPYRIGHT 2001 BIOSIS SO Cell Growth & Differentiation. (1996) Vol. 7, No. 10, pp. to represent the successful identification of a polymorphic phospholipase can provide active tumour resistance. The AN 1996:527044 BIOSIS DN PREV199699249400 trait locus in mammals. ISSN: 1044-9523 **DUPLICATE** 4 Previously, we association of English DT Article 1361-1368.

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TI Method for identifying mutant alleles of mouse affecting a genetic
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                   backcrosses. The mapping is supported by a LOD score exceeding
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD,
                                                                                                                                                                                    a variety of human tumors, including colon tumors. These results
                                                                        Interesting, Mom-1 lies in a region of synteny conservation with
                                                                                                                                                                                                                                       evidence of a major modifier affecting expression of an inherited
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               APPLICATION NO.
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                                                                                                                             chromsome 1p35-36, a region of frequent somatic loss of
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AN 2000:68594 CAPLUS
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           locus and their use in screening for human homologs
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 IN Dove, William F.; Shedlovsky, Alexandra
PA Wisconsin Alumni Research Foundation, USA
SO PCT Int. Appl., 37 pp.
CODEN: PIXXD2
                                                                                                                                                                                                                                                                                                                                                                                                                                                        5586 ETHYLNITROSOUREA/AB,BI
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           1788 L22 AND MUTAGEN%AB.BI
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                                                                                                                                                                                                                                                                                                                                                                           => s ethylnitrosourea/ab,bi
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                                                                                                                                                          heterozygosity in
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LA English
                                                                                                                                                                                                                                                                                              syndrome.
intraspecific
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in two by a LOD score exceeding 14. Interestingly, Mom-1

tumor number

TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, RU, TJ, TM KG, KZ, MD SK, SL, TJ

RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK,

ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG,

CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG 9949843 AI 20000207 AU 1999-49843 19990712 PRAI US 1998-114973 19980714 WO 1999-USI 5661 19990712 AU 9949843

AB A method for breeding ***mutagenized*** mice that permits detection of

genetic loci that can modify a known index phenotype involves

mutagenized founder strain and a second strain of mice allele at a locus that confers the index phenotype. In the test

generation, clusters of individuals are obsd. to deviate from the typical

phenotype. The genetic material and mols, encoded thereby can be using available methods. Improved and compact methods called obtained

(index-directed, cluster-enhanced, ***Modifier***

locus and

ICMM

Molecule identification method) are also disclosed. The method is exemplified by identification of the suppressor or enhancer alleles

mouse Min allele of APC locus by phenotypic and genotypic

generation (and their outcross and backcross offsprings) of ***mutagenized*** ***ethylnitrosourea*** studies of F1

heterozygous B6-APCmin/+ male. The identification of these new the mouse disease models for human colon cancers are helpful to genes in

human homologs involved in the related diseases

RE.CNT 4

Anon; GENETICS 1996, V144(4), P1777

(2) Dietrich, W; "GENETIC IDENTIFICATION OF MOM-1, A

MAJOR MODIFIER LOCUS

AFFECTING MIN-INDUCED INTESTINAL NEOPLASIA IN THE MOUSE" CELL V75, P631

(3) Gould, K; Genetic evaluation of candidate genes for the Moml CAPLUS modifier of

intestinal neoplasia in mice

(4) Wisconsin Alumni Res Found, WO 9822622 A 1998 CAPLUS

=> s 123 and backcross?/ab,bi

7 L23 AND BACKCROSS?//AB,BI AB' IS NOT A VALID FIELD CODE

=> dup rem 125

PROCESSING COMPLETED FOR L25 L26 4 DUP REM L25 (3 DUPLICATES REMOVED)

=> d 1- bib ab

YOU HAVE REQUESTED DATA FROM 4 ANSWERS CONTINUE? Y((N);

L26 ANSWER I OF 4 CAPLUS COPYRIGHT 2001 ACS AN 2000:68594 CAPLUS DN 132:103741

TI Method for identifying mutant alleles of mouse affecting a genetic disease

locus and their use in screening for human homologs

Wisconsin Alumni Research Foundation, USA IN Dove, William F.; Shedlovsky, Alexandra PA Wisconsin Alumni Research Foundation, I SO PCT Int. Appl., 37 pp.

CODEN: PIXXD2

DT Patent

LA English FAN.CNT 1

APPLICATION NO. KIND DATE PATENT NO. DATE WO 1999-US15661 PI WO 2000004186 A1 20000127 W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ

DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS,

JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU. LV, MD, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, MG, MK,

TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, SK, SL, TJ,

RU, TI, TM RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK.

ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ,

AU 1999-49843 19990712 CI, CM, GA, GN, ML, MR, NE, SN, TD, TG AU 9949843 A1 20000207 AU 1999-49843 15 PRAI US 1998-114973 19980714

WO 1999-US15661 19990712

AB A method for breeding ***mutagenized*** mice that permits detection of

genetic loci that can modify a known index phenotype involves crossing a

mutagenized founder strain and a second strain of mice carrying an

allele at a locus that confers the index phenotype. In the test generation, clusters of individuals are obsd. to deviate from the

phenotype. The genetic material and mols, encoded thereby can be

using available methods. Improved and compact methods called

(index-directed, cluster-enhanced, Modifier tocus and Molecule identification method) are also disclosed. The method is exemplified by

identification of the suppressor or enhancer alleles of mouse Min

of APC locus by phenotypic and genotypic studies of F1 generation

ethylnitrosourea ***mutagenized*** female BTBR their outcross and ***backcross*** offsprings) of

heterozygous B6-APCmin/+ male. The identification of these new genes in

the mouse disease models for human colon cancers are helpful to

human homologs involved in the related diseases

RE.CNT 4

(1) Anon; GENETICS 1996, V144(4), P1777

(2) Dietrich, W., "GENETIC IDENTIFICATION OF MOM-1, A MAJOR MODIFIER LOCUS

AFFECTING MIN-INDUCED INTESTINAL NEOPLASIA IN THE MOUSE" CELL V75, P631 CAPLUS

Gould, K.; Genetic evaluation of candidate genes for the Morn! modifier of

(4) Wisconsin Alumni Res Found; WO 9822622 A 1998 CAPLUS intestinal neoplasia in mice

L26 ANSWER 2 OF 4 MEDLINE AN 2000409356 MEDLINE DN 20344604

TI Cryoconservation--archiving for the future.

AU Glenister P.H.; Thomton C.E.
CS. MRC Mammalian Genetics Unit, Harwell, Oxon OX11 ORD, UK...

SO MAMMALIAN GENOME, (2000 Jul) 11 (7) 565-71. Ref: 53 P.Glenister@har.mrc.ac.uk

Journal code: BES, ISSN: 0938-8990.

CY United States
DT Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW) (REVIEW, TUTORIAL)

LA English FS Priority Journals

EW 20001101

AB Mouse genetics is set to play a pivotal role in the key

challenge-the study of mammalian gene function. Addressing this

'AB' IS NOT A VALID FIELD CODE 127 39 L9 AND BACKCROSS/// hyperphenylalaninemia in man. => s 19 and backcross?/ab,bi LA English FS Priority Journals United States ***backcross*** Micro-pedigree EM 198808 induced by leading C.Y ΑB for 2 space. Cryopreservation of both mouse embryos and spermatozoa is skin in the interscapular region. In contrast to the Tsk mutation (on current cryopreservation approaches. Comprehensive mouse mutant Moreover, frozen oocytes and ovaries may offer a valuable addition ***ethylnitrosourea*** . The mouse was recognized because of archives of frozen spermatozoa provides a potential powerful route Tsk2/+ is a novel mutation that first appeared in the offspring of genome that contains the Tsk2 mutation. Thus, the position of the mutation cosegregates with 4 microsatellite markers and with gene L26 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2001 ACS AN 1996:550637 CAPLUS DN 125:187267 TI A high-resolution linkage map of the tight skin 2 (Tsk2) locus: a model for scleroderma (SSc) and other cutaneous fibrotic diseases J Christner, P.J.; Siracusa, L.D.; Hawkins, D.F.; McGrath, R.; mouse from the 101/H strain that was ***mutagenized*** with The authors report the results of intraspecific and intersubspecific ***backcross*** studies performed to define the minimal and dissemination of new and existing mouse strains is simplified will involve the development and application of systematic
mutagenesis approaches. The expanding mouse mutant availability of extensive frozen archives. Also, the availability of mutation was localized to the proximal region of chromosome 1. chromosome 2), the Tsk2 mutation has been localized to mouse will result threatens to overwhelm the currently available animal widely employed for the efficient archiving of mouse stocks. production of ***backcross*** progeny for rapid genetic Ball, S.T.; Jimenez, S.A.; Peters, J. CS. Jefferson Medical College, Thomas Jefferson University, Philadelphia, PA, will provide an essential resource for mammalian genetics SO Mamm. Genome (1996), 7(8), 610-612 CODEN: MAMGEC; ISSN: 0938-8990 21(st) century. 19107, USA DT Journal Distribution region of the currently ۲

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recessive mutations that cause defects in phenylalanine metabolism
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                                                                                                                                                                                                                                                                                    DUPLICATE 1
and is flanked on the proximal side by D1Mit233 and on the distal
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 Division of Biology, Kansas State University, Manhattan 66506.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       cells and a three-generation breeding scheme were used to screen
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       assignment. The initial symptomatology of the mutant phenotype
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             to elevated serum levels of this amino acid. This paper describes
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          this mutant may represent a useful animal model for the study of
                                                                                                                                                                                                                                                                           L26 ANSWER 4 OF 4 MEDLINE
AN 88196848 MEDLINE
DN 88196848
TI hph-1: a mouse mutant with hereditary hyperphenylalaninemia
                                                                                      DIMit213. These markers reside <1cM apart on the published
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             very near Np-1 and a ***backcross*** experiment with a
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  mapping experiment places the mutant gene locus on mouse
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                inbred mouse strain involving a nearby locus confirms the
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        affected and unaffected animals indicate that the mutation
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        ***Ethylnitrosourea*** ***mutagenesis*** of
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   an autosomal recessive manner. An interspecies mouse
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      effectively clear a phenylalanine challenge in the adult.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                            isolation of such a mutation, hph-1, causing a heritable
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         AU Bode V.C., McDonald J.D., Guenet J.L.; Simon D. CS. Division of Biology, Kansas State University, Man NC 5 R01 HD5354-06 (NICHD)
SO. GENETICS, (1988 Feb) 118 (2) 299-305.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             ***ethylnitrosourea*** ***mutagenesis***
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          Journal; Article; (JOURNAL ARTICLE)
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       Journal code: FNH. ISSN: 0016-6731.
                                                                                                                                                                                         linkage map for mouse chromosome 1
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=> s 127 and mutagen?/ab,bi
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'AB' IS NOT A VALID FIELD CODE L28 2 L27 AND MUTAGEN?/AB,BI

=> dup rem 128

PROCESSING COMPLETTED FOR L28 2 DUP REM L28 (0 DUPLICATES REMOVED)

=> d 1- bib ab

YOU HAVE REQUESTED DATA FROM 2 ANSWERS CONTINUE? Y/(N):y

L29 ANSWER I OF 2 CAPLUS COPYRIGHT 2001 ACS AN 2000:68594 CAPLUS

TI Method for identifying mutant alleles of mouse affecting a genetic DN 132:103741

locus and their use in screening for human homologs

PA Wisconsin Alumni Research Foundation, USA SO PCT Int. Appl., 37 pp. IN Dove, William F.; Shedlovsky, Alexandra

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

APPLICATION NO. KIND DATE PATENT NO. DATE WO 1999-USI 5661 PI WO 2000004186 AI 20000127

19990712

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ

DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, II., JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, IN, IS,

MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ MG, MK,

TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY RU, TJ, TM KG, KZ, MD,

RW GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT. BE, CH, CY, DE, DK,

AU 1999-49843 19990712 ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG A1 20000207 AU 9949843

WO 1999-US15661 19990712

PRAI US 1998-114973 19980714

AB A method for breeding ***mutagenized*** mice that permits

genetic loci that can modify a known index phenotype involves

Molecule identification method) are also disclosed. The method is phenotype. The genetic material and mols, encoded thereby can be conductance regulator deficient mice by a secondary genetic factor exemplified by identification of the suppressor or enhancer alleles generation (and their outcross and ***backcross*** offsprings) B6-APCmin/+ male. The identification of these new genes in the ***mutagenized*** founder strain and a second strain of mice AFFECTING MIN-INDUCED INTESTINAL NEOPLASIA IN Modulation of disease severity in cystic fibrosis transmembrane CS Department of Molecular Genetics, The University of Toronto. using available methods. Improved and compact methods called (4) Wisconsin Alumni Res Found; WO 9822622 A 1998 CAPLUS published erratum appears in Nat Genet 1996 May;13(1):129] (2) Dietrich, W., "GENETIC IDENTIFICATION OF MOM-1, A Gould, K.; Genetic evaluation of candidate genes for the Mornl generation, clusters of individuals are obsd. to deviate from the AU Rozmahel R; Wilschanski M; Matin A; Plyte S; Oliver M; disease models for human colon cancers are helpful to screen mouse Min allele of APC locus by phenotypic and genotypic allele at a locus that confers the index phenotype. In the test ethylnitrosourea ***mutagenized*** female BTBR and A; Forstner J; Durie P; Nadeau J; Bear C; Tsui L C (index-directed, cluster-enhanced, ***Modifier*** SO NATURE GENETICS, (1996 Mar) 12 (3) 280-7 Journal; Article; (JOURNAL ARTICLE) Anon; GENETICS 1996, V144(4), P1777 homologs involved in the related diseases Journal code: BRO, ISSN: 1061-4036 L29 ANSWER 2 OF 2 MEDLINE AN 96172827 MEDLINE DN 96172827 TI Modulation of disease severity in THE MOUSE" CELL V75, P631 MAJOR MODIFIER LOCUS intestinal neoplasia in mice DT Journal; Article; (, LA English FS Priority Journals EM 199605 Auerbach W, Moore United States ***locus*** and RE.CNT 4 obtained ζ

Identification of modifier genes in our Cftr(m1HSC)/Cftr(m1HSC) ***backcross*** and intercross progeny with different inbred conductance regulator (Cftr) usually die of intestinal obstruction should provide important insight into the heterogeneous disease in part by up-regulation of a calcium-activated CI- conductance rectification of CI- and Na+ ion transport abnormalities can be studies on mice with prolonged survival show that the partial have created Cftr-deficient mice and demonstrate prolonged AB Mice that have been made deficient for the cystic fibrosis suggesting that modulation of disease severity is genetically A genome scan showed that the major ***modifier*** maps near the centromere of mouse chromosome 7. presentation observed among CF patients. Electrophysiological survival among ***locus determined strains,

PROCESSING COMPLETED FOR L27
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L30 ANSWER I OF 13 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE I
AN 2001-151886 BIOSIS
DN PREVZO0100151886
IT Epistatic interactions between skin tumor *modifier***
****loci****

in interspecific (spretus/musculus) ***backcross*** mice. AU Nagase, Hiroki; Mao, Jian-Hua; de Koning, John P.; Minami, Tomoe, Balmain,

Allan (1)
CS (1) University of California-San Francisco Comprehensive
Cancer Center,

2340 Sutter Street, San Francisco, CA, 94143 USA SO Cancer Research, (February 15, 2001) Vol. 61, No. 4, pp. 1305-1308, print. ISSN: 0008-5472.

DT Article LA English

SL English
AB The development of cancer is influe

AB The development of cancer is influenced both by exposure to environmental carcinogens and by the host genetic background. Epistatic

interactions between genes are important in determining phenotype in plant and animal

systems and are likely to be major contributors to cancer ascentibility

in humans. Several tumor ***modificr*** ***loci*** have een

identified from studies of mouse models of human cancer, and genetic

interactions between ***modifier*** ***loci*** have been detected by genome scarning using recombinant congenic strains of mice (R.

of performant et al., Nat. Genet., 14: 465-467, 1996; T. van Wezel et al., Nat.

14: 468-470, 1996; W. N. Frankel et al., Nat. Genet., 14, 371-373, 996)

Genet

We demonstrate here that strong genetic interactions between skin umor
modifier ***loci*** can be detected by hierarchical

••••modifier••• ••••loci*•• can be detected by hierarchical thole
genome scanning of a complete interspecific ••••backcross*••

(outbred

Mus spretus X Mus musculus (NIH/Ola)). A locus on chromosome
7 (Skts1)
showed a highly significant interaction with Skts5 on chromosome

2 (P < 10-16), whereas additional significant interactions were detected

loci on chromosomes 4 and 5, and 16 and 15. Some of these numnitative

trait loci and their interactions, in particular the Skts1-Skts5 interaction, were confirmed in two completely independent ***ebackcrosses*** using inbred spretus strains (SEGOPas and ***Petrffi;)

and NIH/Ola. These results, therefore, illustrate the general use of interspecific crosses between Mus musculus and Mus spretus for

detection of strong genetic interactions between tumor modifier

L30 ANSWER 2 OF 13 MEDLINE DUPLICATE

AN 2001089278 MEDLINE DN 20565764 TI Efficiency alleles of the Pctrl ***modifier*** ***locus****

for

plasmacytoma susceptibility.

AU Zhang S L; DuBois W; Ramsay E S; Bliskovski V; Morse H C; Taddesse-Heath

L; Vass W C; DePinho R A; Mock B A CS Laboratory of Genetics, Division of Basic Sciences, National

Cancer Institute, National Institutes of Health, Bethesda, Maryland 20892,

USA. SO MOLECULAR AND CELLULAR BIOLOGY, (2001 Jan) 21 (1) 310-8.

DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, Molecule identification method) are also disclosed. The method is W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY. mutagenized founder strain and a second strain of mice carrying an AB A method for breeding mutagenized mice that permits detection at a locus that confers the index phenotype. In the test generation, clusters of individuals are obsd. to deviate from the typical available methods. Improved and compact methods called ICMM exemplified by identification of the suppressor or enhancer alleles generation (and their outcross and ***backcross*** offsprings) AU 1999-49843 19990712 loci that can modify a known index phenotype involves crossing a male. The identification of these new genes in the mouse disease MN, MW, MX, NO, NZ, PL. PT. RO, RU, SD, SE, SG, SI, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL. PT. SE, BF, BJ, The genetic material and mols, encoded thereby can be obtained RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, ethylnitrosourea mutagenized female BTBR and heterozygous for human colon cancers are helpful to screen human homologs APPLICATION NO. PI WO 2000004186 AI 20000127 WO 1999-USI 5661 mouse Min allele of APC locus by phenotypic and genotypic CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG 9949843 A1 20000207 AU 1999-49843 (index-directed, cluster-enhanced, ***Modifier*** locus and their use in screening for human homologs IN Dove, William F.; Shedlovsky, Alexandra PA Wisconsin Alumni Research Foundation, USA SO PCT Int. Appl., 37 pp. KIND DATE PRAI US 1998-114973 19980714 WO 1999-USI 5661 19990712 CODEN: PIXXD2 RU, TJ, TM PATENT NO. ***locus*** and AU 9949843 CH, CY, DE, DK KG, KZ, MD LA English FAN.CNT 1 DT Patent CN, CU, C SK, SL, TJ, of genetic MG, MK CF. CG. IN, IS, ğ ō DN 132:103741 TI Method for identifying mutant alleles of mouse affecting a genetic disease respect to their resistant Petr1 counterparts in DBA/2 and C57BL/6 strains are genetically resistant to the plasmacytomagenic effects of traditionally susceptible BALB/cAn strain. Biological assays of the interval. Concomitantly, resistant C57B1/6 mice, from which both alleles displayed similar potencies in both assays. We propose that pristane. In this model system for human B-cell neoplasia, one of (45). In the present study, alleles of Pctr1, Cdkn2a, and D4Mit15 pristane-induced plasma cell tumors over a shorter latency period ***locus*** , Pctrl complex genetic trait involving multiple loci, while DBA/2 and resistant strain (BALB/cDAG) carrying DBA/2 chromatin were C.DAG-Petr1 Cdkn2a D4Mit15 congenic was more resistant to plasmacytomagenesis than BALB/c, thus narrowing Petr1 to a products of the Cdkn2a gene have been eliminated, developed p16(INK4a) and p19(ARF) alleles from BALB/c and DBA/2 ***backcrossed*** to the susceptible BALB/c strain. The BALB/c p16(INK4a) and p19(ARF) alleles were found to be inducing growth arrest of mouse plasmacytoma cell lines and ***[00]*** mapped to a 5.7-centimorgan (cM) chromosomal region that ras-induced transformation of NIH 3T3 cells, while the two L30 ANSWER 3 OF 13 CAPLUS COPYRIGHT 2001 ACS BALB/c p16(INK4a) allele was less active than its DBA/2 which encodes p16(INK4a) and p19(ARF), and the coding AB The susceptibility of BALB/c mice to pristane-induced BALB/c susceptibility and ***modifier*** Journal; Article; (JOURNAL ARTICLE) "efficiency" allele of the p16(INK4a) gene BALB/c susceptibility/ ***modifier*** Journal code: NGY. ISSN: 0270-7306 2000:68594 CAPLUS 132:103741 LA English FS Priority Journals EM 200101 United States plasmacytomas is a

resultant

1.5-cM

indicated that the counterpart in preventing

included Cdkn2a, sequences for the polymorphic with

CS7BL/6

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metallothionein promoter exhibit severe pigmentation of the whole
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        AB Transgenic mice carrying the RET oncogene under the control of
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                TI Mapping of melanoma ***modifier*** ***loci*** in RET
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 DUPLICATE
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                                                                                                                                                                         AFFECTING MIN-INDUCED INTESTINAL NEOPLASIA IN
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                                                               (1) Anon; GENETICS 1996, V144(4), P1777
(2) Dietrich, W; "GENETIC IDENTIFICATION OF MOM-1, A
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                                                                                                                                                                                                                                                                                              (3) Gould, K; Genetic evaluation of candidate genes for the Moml
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 AU Dragani T A; Peissel B; Zanesi N; Aloisi A; Dai Y; Kato M;
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              Venezian Milan, Italy.. dragani@istitutotumori.mi.it
SO JAPANESE JOURNAL OF CANCER RESEARCH, (2000
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   in RET mice; founder mice crossed with BALI3/c mice show
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               CS Department of Experimental Oncology, Istituto Nazionale
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         Journal; Article; (JOURNAL ARTICLE)
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                                                                                                                                                                                                                 THE MOUSE" CELL V75, P631
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                                                                                                                                               MAJOR MODIFIER LOCUS
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the related diseases.

involved in

susceptibility alleles is significantly linked to early mortality caused by rapid carcinoma growth (chi(2) = 25.22; P = $5.1 \times 10(-8)$). These rate and/or survival of individual ***backcross*** mice exposed are close to or the same as loci associated with carcinoma incidence survival of mice with carcinomas, whereas the reverse combination TI Epilepsy in mice deficient in the 65-kDa isoform of glutamic acid unknown, but differences in saturation with cofactor and subcellular indicate that host genetic factors may be used to predict carcinoma AU Kash S F; Johnson R S; Tecott L H; Noebels J L; Mayfield R D; encoding GAD65 and ***backcrossed*** the mutation into the regulation of inhibitory neurotransmission. We have disrupted the same carcinogenic stimulus and suggest that mouse models may strain of mice. In contrast to GAD67-/- animals, which are born CS Department of Medicine, School of Medicine, University of localization suggest that GAD65 may provide reserve pools of and/or papilloma multiplicity, is significantly associated with SO PROCEEDINGS OF THE NATIONAL ACADEMY OF approach to the identification of genetic modifiers of cancer isoforms, GAD65 and GAD67. The separate role of the two AB gamma-Aminobutyric acid (GABA), the major inhibitory the mammalian brain, is synthesized by two glutamate San Francisco, San Francisco, CA 94143, USA CY United States
DT Journal; Article; (JOURNAL ARTICLE) AMERICA, (1997 Dec 9) 94 (25) 14060-5. SCIENCES OF THE UNITED STATES OF Journal code: PV3, ISSN: 0027-8424 FS Priority Journals; Cancer Journals L30 ANSWER 7 OF 13 MEDLINE AN 1998054360 MEDLINE NS29709/11535 (NINDS) NC DK41822 (NIDDK) neurotransmitter in Backkeskov S EW 19980303 DN 98054360 decarboxylase California at LA English EM 199803 Hanahan D, survival in humans. isoforms is provide an GABA for growth DUPLICATE [D6Mit15 (Skts12), D7Mit12 (Skts2), and D17Mit7 (Skts10)], all are potentially important candidate genes which may be linked to AB Studies of mouse models of human cancer have established the (5) Drake, C; Proc Natl Acad Sci USA 1994, V91, P4062 CAPLUS associated with time of survival after development of malignant progression. We have carried out an analysis of skin have identified another seven loci showing either significant (six susceptibility in interspecific Mus musculus/Mus spretus hybrid activities affects the outcome of disease features in the progeny. University of California San Francisco Cancer Center, Cancer specific search was carried out for skin tumor ***modifier*** SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1999 Dec 21) 96 (26) 15032-7. Journal code: PV3. ISSN: 0027-8424. multiple tumor modifiers that influence parameters of cancer malignant tumor. A combination of resistance alleles at three susceptibility such as tumor multiplicity, tumor size, or the or suggestive (one locus) linkage to turnor susceptibility or Institute, University of California, 2340 Sutter Street, San ***|OC!*** ALL CITATIONS AVAILABLE IN THE RE FORMAT (7) Egle, A: Eur J Immunol 1996, V26, P3119 CAPLUS (9) Hirose, S: Int Immunol 1994, V6, P1857 CAPLUS (12) Jiang, Y: J Immunol 1997, V158, P992 CAPLUS (4) Dietrich, W. Genetics 1992, V131, P423 CAPLUS AN 2000079602 MEDLINE
DN 20079602
TI A subset of skin tumor ***modifier*** Journal; Article; (JOURNAL ARTICLE) regulation of AEA and splenomegaly. survival time of tumor-bearing mice. Priority Journals; Cancer Journals L30 ANSWER 6 OF 13 MEDLINE AU Nagase H; Mao J H; Balmain A United States ***loci*** EW 20000305 94105, USA. Francisco, CA English EM 200003 RECONT 31 existence of determines probability mice and CY DI the FS FS Engineering, Toin University of Yokohama, Yokohama, 225-8502, on chromosomes 6, 8, 9, 12, and 13 indicated allelic imbalance in autoantibodies (AEA) in assocn with splenomegaly, thus serving combined effect of two major independently segregating dominant one linked to D7MIT30 on chromosome 7 and the other linked to inherited as a dominant fashion, expression in F1 hybrids of NZB non-New Zealand strains is suppressed due to the contribution of modifying genes present in the latter strains. Using chromosomal chromosome 10. Splenomegaly was modified mainly by a single mice, with a significant excess of BALB/c alleles, suggesting the for autoimmune hemolytic anemia. Although these autoimmune autoimmune hemolytic anemia-prone New Zealand Black mice Ochiai, Kimiko; Ozaki, Shoichi; Tanino, Akihiro; Watanabe, CS Toin Human Science and Technology Center, Department of microsatellite markers in the (C57BL/6 times, NZB)F1 times Tomoo; Mitsui, Kenichi; Toei, Junichi; Inada, Yuji; Hirose, ***backcross*** progeny, the authors mapped C57BL/6 L30 ANSWER 5 OF 13 CAPLUS COPYRIGHT 2001 ACS anemia in the NZB strain is under multigenic control and a Genetic regulation of anti-erythrocyte autoantibodies and New Zealand Black (NZB) mice spontaneously produce AEA prodn. and splenomegaly. Generation of AEA was of not only susceptibility but also modifying alleles with allele linked to D4MIT58 on chromosome 4. Thus, the of additional putative melanoma ***modifier*** Shirai, Toshikazu; Nishimura, Hiroyuki Int. Immunol. (2000), 12(1), 1-8 CODEN: INIMEN; ISSN: 0953-8178 PB Oxford University Press DT Journal LA English AB New Zealand Black (NZB) 2000:80654 CAPLUS autoimmune hemolytic down-regulated by a modifying loci for chromosomes. 132:235822 splenomegaly in combined action anti-erythrocyte D10MIT42 on Shinji; Ueno, Biomedical as a model traits are and most presence

DUPLICATE

chromosome 17 in moderate and low penetrance crosses of CT with Identification of modifier genes in our Cftr(m1HSC)/Cftr(m1HSC) and Mus spretus. No effect of mct1 was seen in a higher penetrance AB The major gene for neural tube defects, ct, in the curly-tail (CT) DUPLICATE modifiers or strain-specific susceptibility alleles. Here we describe ***locus***, mctl, on mouse mapping of a curly-tail ***modifier*** ***locus*** , mctl, ***backcross*** and intercross progeny with different inbred AU Letts V A; Schork N J; Copp A J; Bernfield M; Frankel W N CS Jackson Laboratory, Bar Harbor, Maine 04609, USA. NC HD28882 (NICHD)
SO GENOMICS, (1995 Oct 10) 29 (3) 719-24. trait, already incomplete in the CT strain, was further reduced in conductance regulator (Cftr) usually die of intestinal obstruction. should provide important insight into the heterogeneous disease data from several ***backcrosses*** . The penetrance of the in part by up-regulation of a calcium-activated CI- conductance rectification of CI- and Na+ ion transport abnormalities can be studies on mice with prolonged survival show that the partial have created Citr-deficient mice and demonstrate prolonged suggesting that modulation of disease severity is genetically of these ***backcrosses***, suggesting the existence of strain was mapped previously to mouse chromosome 4 by A genome scan showed that the major ***modifier*** maps near the centromere of mouse chromosome 7. Journal; Article; (JOURNAL ARTICLE) presentation observed among CF patients. Journal code: GEN, ISSN: 0888-7543, L30 ANSWER 9 OF 13 MEDLINE Tl A curly-tail ***modifier*** chromosome AN 96121384 MEDLINE LA English FS Priority Journals Electrophysiological CY United States combining linkage survival among 96121384 EM 199605 determined. BALB/cByJ neural tube explained recessive Ω̈́ result in increased mortality. Seizures can be precipitated by fear or the brain. These data suggest that GABA synthesized by GAD65 is mice ***backcrossed*** into a second genetic background, the developmental abnormalities and die shortly after birth, GAD65-/appear normal at birth. Basal GABA levels and holo-GAD activity diabetic (NOD/LtJ) strain of mice enabling electroencephalogram in the dynamic regulation of neural network excitability, implicate DUPLICATE conductance regulator deficient mice by a secondary genetic factor of the seizures. The generally higher basal brain GABA levels in suggesting that the relative contribution of GABA synthesized by Modulation of disease severity in cystic fibrosis transmembrane ***backcross*** are significantly decreased by the GAD65-/-CS Department of Molecular Genetics, The University of Toronto, published erratum appears in Nat Genet 1996 May;13(1):129] mild stress. Seizure susceptibility is dramatically increased in c-fos-like immunoreactivity reveals the involvement of limbic significantly decreased. GAD65-/- mice develop spontaneous least one ***modifier*** ***locus*** in the NOD/LtJ AU Rozmahel R; Wilschanski M; Matin A; Plyte S; Oliver M; normal, but the pyridoxal 5' phosphate-inducible apo-enzyme LA English FS Priority Journals EM 199605 AB Mice that have been made deficient for the cystic fibrosis present GAD65-/- animals as a model of epilepsy involving A; Forstner J; Durie P; Nadeau J; Bear C; Tsui L C SO NATURE GENETICS, (1996 Mar) 12 (3) 280-7. Journal code: BRO. ISSN: 1061-4036. total brain GABA levels is genetically determined. Journal; Article; (JOURNAL ARTICLE) L30 ANSWER 8 OF 13 MEDLINE AN 96172827 MEDLINE DN 96172827 TI Modulation of disease serve Auerbach W; Moore United States Seizure-associated GAD65-/regions of strain, and 겁

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Tl Steroid sulfatase and the Y chromosome hypertensive locus of the
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               spontaneously hypertensive rat.

AU Johnson M.L.; Ely D.L.; Turner M.E.
CS Midwest Hypertension Research Center, Omaha, Nebraska, USA
SO STEROIDS, (1995 Oct) 60 (10) 681-5.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   conversion of steroid sulfates to their active nonconjugated form. In
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                                                                                                                                                                                                                            overall. No evidence was found for epistatic interaction between ct
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                                                                      all of the affected individuals in the BALB/cBy1 cross and most of
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                increases blood pressure. This locus requires an androgen receptor
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     STS activity in testes, adrenal gland, liver, and hypothalamus. The
                                                                                                                                               affected individuals in the M. spretus cross and was the preferred
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           values for STS in the two strains were not significantly different;
model. Homozygosity at both ct and mct1 loci was sufficient to
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      the rat Sts is on the X chromosome. We measured STS activity
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          to test and/or confirm a Y chromosome influence on STS. STS
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   between SHR and WKY, the STS activity could be a secondary
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             testosterone for maximum expression. Steroid sulfatase (STS)
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    activity differences were likely due to differences in enzyme
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          Because the blood pressures of SHR/y and SHR/a were also
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                            and normotensive Wistar Kyoto (WKY) males. SHR had
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        Journal; Article; (JOURNAL ARTICLE)
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              Journal code: V10, ISSN; 0039-128X.
                                                                                                                                                                                                                                                                                                                                                                              L30 ANSWER 10 OF 13 MEDLINE
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FS Priority Journals
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                                                                                                                                                                                                                                                                                                     mct].
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                                                                                                                                                                                          model
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DUPLICATE

with the BXD-8/Ty strain, confirming that ct is the major gene in

loss of heterozygosity in a variety of human tumors, including colon II The Min (multiple intestinal neoplasia) mutation: its effect on gut of multiple intestinal adenomas throughout the duodenal-to-colonic Preliminary ***backcross*** analysis is consistent with a single Paneth cells. Expression of endogenous marker genes within these by the adenoma along the duodenal-to-colonic axis and mirrors the fully penetrant in hybrids with either AKR/1 or MA/MyJ. However, were sought in which Min/+ animals could survive for up to 300 d. immunocytochemical studies of these lesions demonstrate patches AU Moser A R; Dove W F; Roth K A; Gordon J I
CS McArdle Laboratory, University of Wisconsin, Madison 53706.
NC CA07075 (NCI) JOURNAL OF CELL BIOLOGY, (1992 Mar) 116 (6) 1517-26. differentiated enterocytes, and scattered enteroendocrine, goblet To study the time-dependent properties of these tumors, genetic suggests that tumorigenesis in Min/+ mice may be initiated in a differentiated cells can be directly correlated with the position multipotent stem cell normally located at the base of intestinal AB Min is a fully penetrant dominant mutation that leads to the differentiation of the normal gut epithelium. The presence of epithelial cell differentiation and interaction with a modifier tumors. These results provide evidence of a major modifier lineages in adenomas together with their retention of spatial Min/+ C57BL6/J mice have an average life-span of 120 d. hybrids demonstrate a reduction in the number of intestinal expression of an inherited cancer syndrome Journal; Article; (JOURNAL ARTICLE) Journal code: HMV, ISSN: 0021-9525. L30 ANSWER 12 OF 13 MEDLINE Priority Journals; Cancer Journals AN 92176249 MEDLINE CA23076 (NCI) CA50585 (NCI) United States frequent somatic 92176249 LA English FS Priority Jo EM 199206 development information Multi-label conditions affecting occupied multiple SO ŏ hypertension. An alternative hypotheses is that a regulatory locus in and this regulatory locus is on the rat Y chromosome. Further study ***loci*** remain a potential component of the Y chromosome excellent model for familial colon cancer: it carries a mutant mouse the genetic mapping of a locus that strongly modifies tumor number hypothesis can be eliminated, the Sts locus or its ***modifier*** DUPLICATE AB Mutations in the human APC gene caused various familial colon LOD score exceeding 14. Interestingly, Mom-1 lies in a region of tumor phenotype is dramatically modified by genetic background. AU Dietrich W F; Lander E S; Smith J S; Moser A R; Gould K A; Min/+ animals. This gene, Mom-1 (Modifier of Min-1), maps to needed to discriminate between these possibilities, and until the gene and develops many intestinal adenomas. Here, we analyze CS Whitchead Institute for Biomedical Research, Massachusetts addition to the structural locus is responsible for STS activity chromosome 4 and controls about 50% of genetic variation in Genetic identification of Mom-1, a major ***modifier*** conservation with human chromosome 1p35-36, a region of in two intraspecific ***backcrosses*** The mapping is syndromes. The Multiple intestinal neoplasia (Min) mouse affecting Min-induced intestinal neoplasia in the mouse. Journal; Article; (JOURNAL ARTICLE) CELL, (1993 Nov 19) 75 (4) 631-9. Journal code: CQ4, ISSN: 0092-8674 L30 ANSWER 11 OF 13 MEDLINE Priority Journals; Cancer Journals Technology, Cambridge 02142 AN 94061981 MEDLINE Borenstein N; Dove W NC HG00098 (NHGRI) HG00126 (NHGRI) CA07075 (NCI) United States 9406198 supported by a turnor number *** locus hypertensive English 8 Institute of provides an Luongo C; We report ဝွ CY FS EM Å.

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adenomas appear to be established by 100 d of age or sooner. These
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   genetic model. Contrary to expectations, estimates for grain yield of
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                differentiation in the continuously renewing gut epithelium and for
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    cross, and A Composite .times. B Composite. Estimates of half-sib
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             grain moisture and plant and ear heights were greater in the F2 than
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             AU BERNARDO R, JOHNSON G R, DUDLEY J W, MEGHJI M
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         times. Mol 7H was considered. Estimates of genetic parameters in
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              performance (most favorable to least favorable) for all traits being
                                                                                                                                                                  associated with the development of invasive tumors. New tumors
                                                                           MA/MyJ strains. The increased lifespan of the hybrid animals is
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                            times. B Composite]F2.times. [Mol 7H.times. A Composite]F2
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          Introgression of broadbase germplasm to improve the clite single
***modifier*** ***locus*** unlinked to Min in both the
                                                                                                                                                                                                                                                     arise continuously over the lifespan of these animals; instead all
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      assessment of the multi-step hypothesis of intestinal neoplasia.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      L30 ANSWER 13 OF 13 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1990:43589 BIOSIS
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             AB Further improvement in the performance of elite maize (Zea
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              times. A Composite)]BC1 Design 2 population were obtained.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              TI EVALUATION OF F-2 X F-2 AND BC-1 X BC-1 MAIZE
                                                                                                                                                                                                                                                                                                                                                                             indicate that the Min/+ mouse is a powerful model system for
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                hybrids is an important objective of maize breeding programs.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               population and a [B73(B73 times. B Composite]BC1 times.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          covariances (CovHS) and specific combining ability variance
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    CS DEP. AGRON., UNIV. ILL., 1102 S. GOODWIN AVE.,
                                                                                                                                                                                                                                                                                                                                                                                                                                                                    mechanisms that establish and maintain a balance between
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                            the BC1 interpopulation cross as expected for a one-locus,
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          times. Mol 7H, the BC1 interpopulation cross, the F2
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   SO CROP SCI, (1989) 29 (6), 1377-1381
CODEN: CRPSAY ISSN: 0011-183X.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               INTERPOPULATION CROSSES
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              URBANA, ILL. 61801.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        [Mol 7H(Mol 7H
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              proliferation and
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          FS BA; OLD
                                                                                                                                                                                                                                                                                                                                                                                                                                  analyzing the
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           Proportion of
                                         AKR/J and
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   cross B73
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      a [B73 73
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          mays L.)
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                                                                                                                                                                                                           DUPLICATE
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SINCE FILE TOTAL STN INTERNATIONAL LOGOFF AT 17:01:21 ON 02 APR 2001 149.64 153.29 13 DUP REM L27 (26 DUPLICATES REMOVED) DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) 7 S L23 AND BACKCROSS?/AB.BI 4 DUP REM L25 (3 DUPLICATES REMOVED) 39 S L9 AND BACKCROSS?/AB.BI 2 DUP REM L28 (0 DUPLICATES REMOVED) SESSION SESSION 5586 S ETHYLNITROSOUREA/AB,BI 1788 S L22 AND MUTAGEN?/AB,BI 2 S L27 AND MUTAGEN?/AB,BI ENTRY ENTRY FULL ESTIMATED COST 1 S L 23 AND L9 Executing the logoff script... CA SUBSCRIBER PRICE COST IN U.S. DOLLARS TOTAL ---Logging off of STN---SINCE FILE => LOG Y L22 L23 L25 L25 L27 L27 L29 L29 populations for selection procedures that exploit specific combining probability for immediate derivation of superior single crosses from are homozygous for an allele at a ***modifier*** ***locus*** CovHS in the population related to B73 and of VarSCA were two ability efects. Together with higher frequencies of BC1 .times. BC1 testcrosses superior to B73. times. Mol 7H, they indicated a higher cross were consistent with expectations for a model in which B73 times greater, respectively, in the BC1 than in the F2. The large 108 S MODIFIER LOCUS OR MODIFIER LOCI/AB, BI of the modifier allele in the two composites would be near zero FILE 'MEDLINE, EMBASE, BIOSIS, INPADOC, CAPLUS' FILE 'MEDLINE' ENTERED AT 16:41:53 ON 02 APR 2001 which causes complete dominance at a quantitative trait locus 39 S L11 AND BACKCROSS//AB,BI 13 DUP REM L12 (26 DUPLICATES REMOVED) (FILE HOME' ENTERED AT 16:41:46 ON 02 APR 2001) estimates of CovHS and VarSCA for grain yield in the BC, 88 S E1-E4 12 S L11 AND (L19 OR L14) 7 DUP REM L20 (5 DUPLICATES REMOVED) 12 S L14 AND L3 7 DUP REM L15 (5 DUPLICATES REMOVED) model. The VarSCA results suggested the use of L1 0 S CONGENIC AND MUTAGENESIS MAPPING/AB,BI 3 S MUTAGENESIS MAPPING/AB, BI E SHEDLOVSKY ALEXANDRA/AU 4 S L4 AND MAP?/AB.BI E DOVE WILLIAM F/AU 484 S L8 OR L9 OR L10 ENTERED AT 16:46:21 ON 02 0 S L 14 AND L 10 0 S L 14 AND L 2 0 S L3 AND L2 133 S E2-E3 62 S L4 42 S L 2 442 S L3 ***backcross*** 0.S.L.I interpopulation APR 2001 and Mo17H under this => d his 2 % 2 3 E 22 E 2 L14 L15 L16 L17 L13 22222